A PRACTICAL GUIDE TO DIAGNOSIS

Bovine neonatal diarrhoea

MSD
Animal Health

PREFACE

Neonatal diarrhoea is one of the main calf diseases worldwide, with large economic losses for both dairy and beef cattle. The costs are not only the very well known short-term ones associated with treatment and mortality, but also the negative impact on future productivity such as breeding and the lengthy recovery period calves require to return to normal health.

The rapidly evolving field of neonatal health has moved towards a more integrated approach following the lines of the Herd Health principles. Today, we know the aetiology of neonatal diarrhoea is a complex one involving an interaction between enteropathogens (viruses, bacteria and/or parasites), the calf and environmental factors. In the case of infectious diarrhoea, the presence of enteropathogens is necessary though it is not always sufficient on its own to cause the disease. Usually additional environmental and/or animal risk factors are also present, resulting in the calf becoming diarrhoeaic. Indeed, neonatal diarrhoea is a typical production disease in which diagnosis can often not be established from only taking a microbiological point of view.
The management and prophylactic practices play a primary role in disease prevention. Despite this it is essential to have a microbiological assessment as part of the diagnostic approach to diarrhoea control and prevention.

To ensure an accurate diagnosis that leads to successful treatment and prophylaxis, it is necessary to join epidemiology, clinical examination, necropsy findings and laboratorial results as different pieces of the same puzzle. We have held many meetings with practitioners in Europe and Latin America on neonatal diarrhoea to promote best practice and one of the most frequent questions raised is: ' What is the best method for establishing an accurate diagnosis'. Taking this into consideration, we have tried to show in the course of this guide the most up to date information on diagnosis of neonatal diarrhoea in a visual way, through the use of abundant illustrations and schemes. The aim of this guide is to provide the practitioner with an effective tool to understand, identify and solve neonatal diarrhoea problems on farms.

This book is the second in the series following the very successful: "Bovine respiratory pathology a practical guide to diagnosis", by using both books it should be possible to have information on the main infectious diseases affecting calves worldwide.

Profesor J.V. González Martín (Animal Medicine and Surgery Dept., Veterinary Faculty of Madrid - UCM; Dipl. ECBHM) and Dr. L. Elvira Partida TRIALVET Veterinary Consultancy and Research, Madrid, Spain.

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Dr. Filippini

We would like to thank Dr. Susana Astiz and Nati Pérez-Villalobos for their advise and assistance during this project and whenever we undertake a new venture, and especially Susana for her great job with our previous Neonatal Diarrhea Book; and of course also to our farmers, and their calves, which constitute the main source of information for developing this booklet.

We would like to thank our colleagues Dr. G. Valla, B. Cowley and I. Anderson for their considerable advice and critical proof reading.

Dr. habil B. Makoschey
Global Technical Director Ruminant Biologicals
MSD Animal Health

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a.1. Prevalence of neonatal diarrhoea

Causes of mortality in neonatal calves

MORTALITY (%)

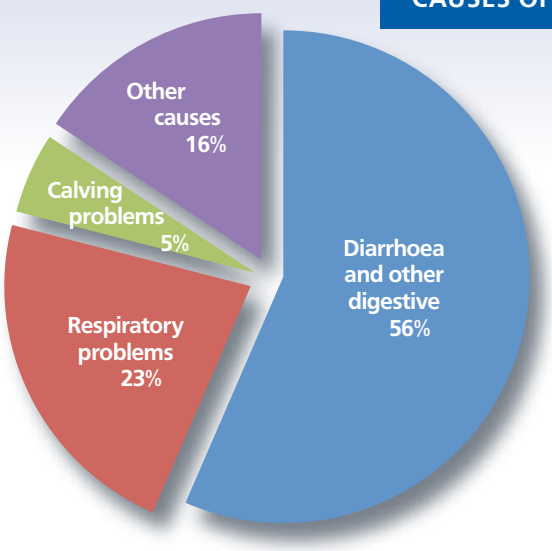
DAIRY CATTLE

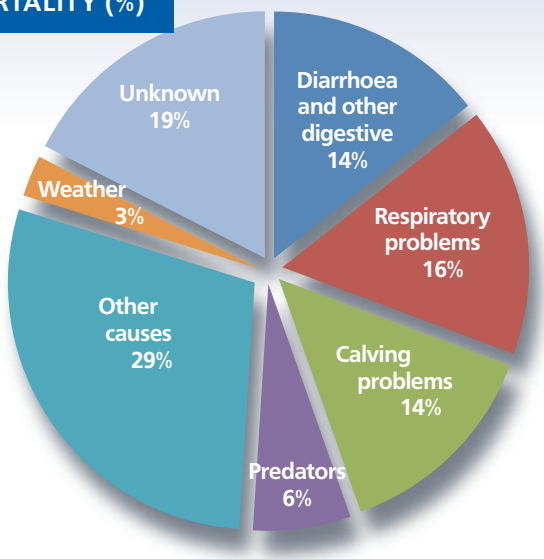
7.8 in USA [1]

BEEF CATTLE

2.3 in USA [2]
11.8 in Canada [3]

CAUSES OF MORTALITY (%)





75% of the mortality of dairy heifers during rearing occurs in the first month of life

50-89% of beef calf mortality occurs in calves younger than one month [2,3]

Between 24 h and 3 weeks of life, 75% of mortality is due to diarrhoea [4]

Within the first month of life, 26% of mortality is due to diarrhoea [3] ;
Diarrhoea is a major cause of neonatal mortality [2]

This indicates the importance of health management systems focused on diarrhoea prevention in newborn calves

a.2. Risk of developing diarrhoea throughout the various calf's stages of life

- In dairy calves, the risk of developing diarrhoea varies with time, with higher cumulative risk of diarrhoea occurring during the first two months of life. Peak risk occurs during the second week of life [5,6]. However, mortality is higher during the first week of life [5].

- In contrast, the period of highest risk of developing diarrhoea occurs earlier in beef calves - during the first week of life [7].

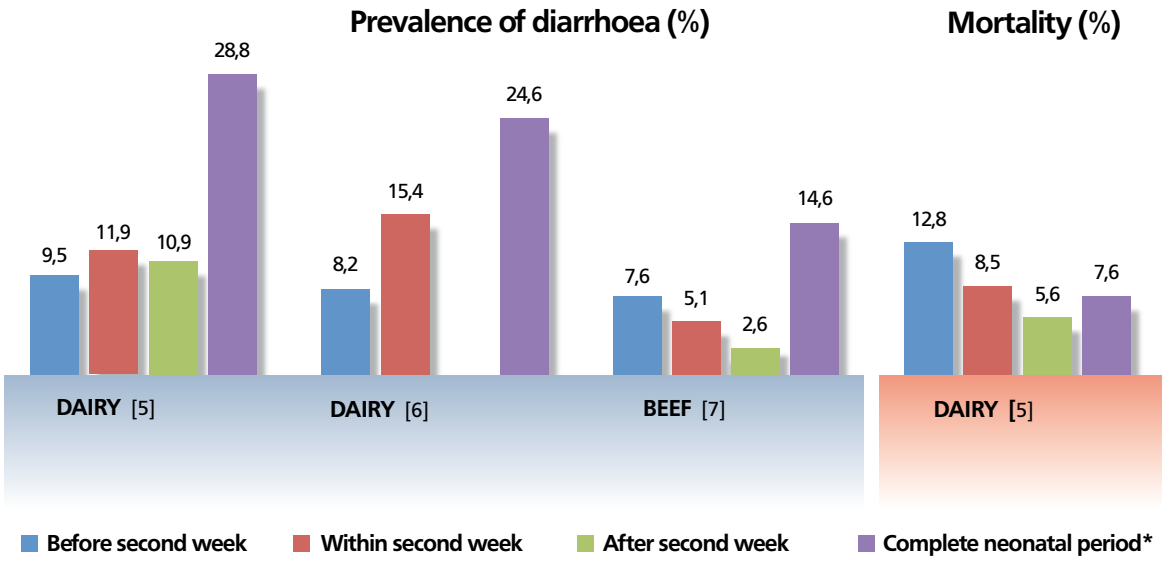
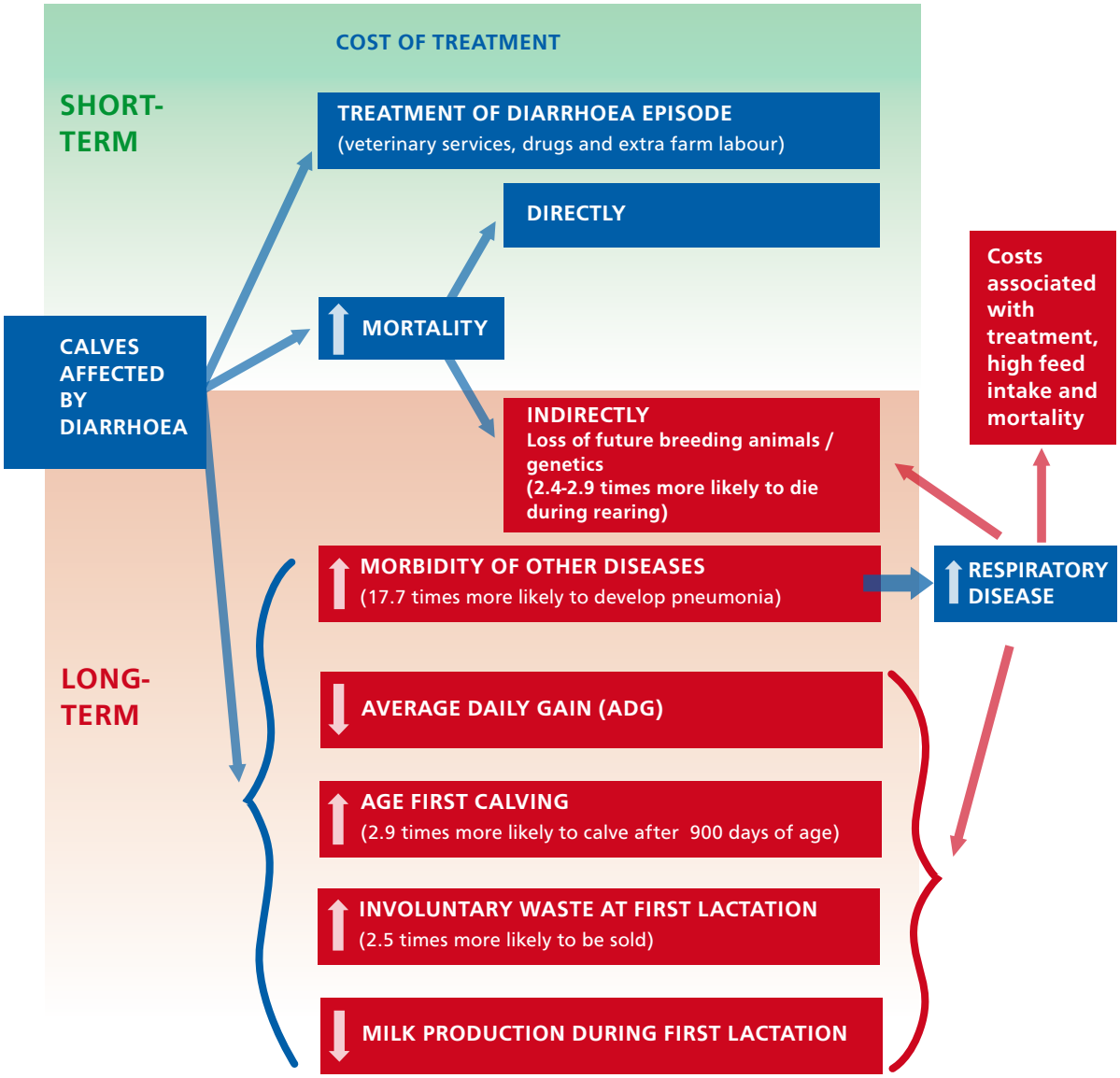


Figure. Left: Prevalence of diarrhoea throughout the calf's stages and cumulative risk over the entire neonatal period.
Right: Prevalence of mortality throughout the calf's stages and average over the entire neonatal period.
*Length of neonatal period: < 3 months [5], < 2 months [6] , < 1 month [7].

a.3. Effect of neonatal diarrhoea

Short- and long-term effects associated with diarrhoea in calves [5,8-20]



a.4. Cost of diarrhoea on herd replacement

- If we assume that on a farm with a calving interval of 12 months, 50% of calves born are females and 75% of the female calves will survive until first calving, this leaves 38 breeding heifers available per 100 cows per year [4].

- However, as we can see in the table below in farms with increased neonatal morbidity and mortality due to diarrhoea, the number of breeding heifers available per 100 cows is below this number. Since diarrhoea in the heifer delays calving by an average of 1.3 months, the number of heifers calving per year will reduce [9].

Cost of morbidity and mortality due to diarrhoea on annual numbers of replacement heifers per 100 cows

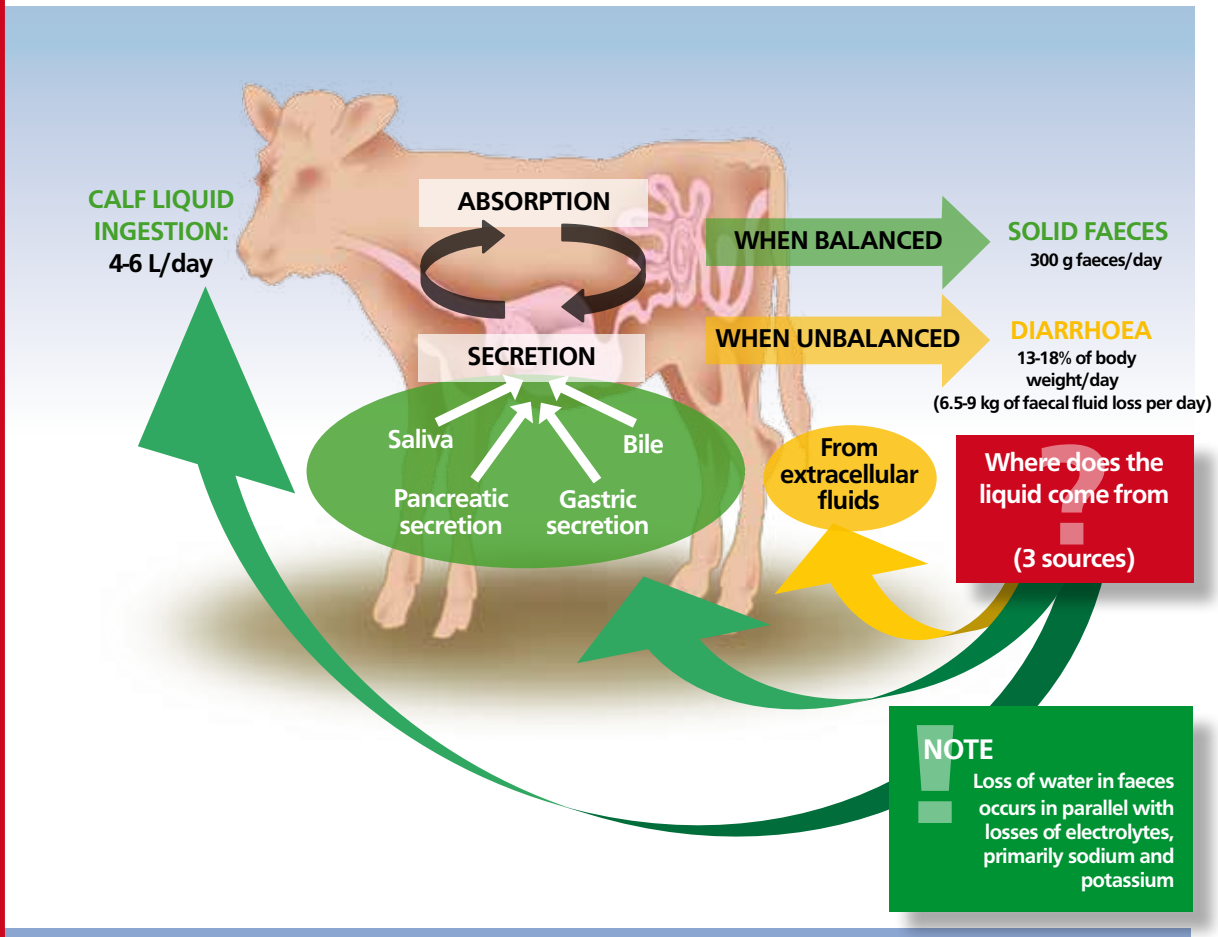
	DIARRHOEA MORTALITY 0%				DIARRHOEA MORTALITY 10%				DIARRHOEA MORTALITY 20%			
	Due to mortality	Due to morbidity	**Total**	**Estimated loss (€)***	Due to mortality	Due to morbidity	**Total**	**Estimated loss (€)***	Due to mortality	Due to morbidity	**Total**	**Estimated loss (€)***
Morbidity 0%	0	0	**0**	**0**	-3.8	0	**-3.8**	**-5,700**	-7.6	0	**-7.6**	**-11,400**
Morbidity 20%	0	-0.4	**-0.4**	**-600**	-3.8	-0.4	**-4.2**	**-6,300**	-7.6	-0.4	**-8.0**	**-12,000**
Morbidity 40%	0	-0.8	**-0.8**	**-1,200**	-3.8	-0.8	**-4.6**	**-6,900**	-7.6	-0.8	**-8.4**	**-12,600**
Morbidity 80%	0	-1.7	**-1.7**	**-2,550**	-3.8	-1.7	**-5.5**	**-8,250**	-7.6	-1.7	**-9.3**	**-13,950**

*To estimate the loss, we have taken into consideration that the price of one prefresh heifer is approximately 1,500 €, so each tenth of a heifer value corresponds to 150 €.

b.1. Pathophysiology of diarrhoea

b.1.1. Definition

At the intestinal level, variations in the balance between absorption and secretion mechanisms will affect faecal water content, resulting in normal faeces when both mechanisms are balanced or in diarrhoeaic faeces when they are unbalanced.



● What is diarrhoea?

Diarrhoea is an increase in the water content, frequency, and volume of faeces.
(Normal faeces is 25% solid material, while in diarrhoea more than 80% of total faeces is water).

● Where does the liquid come from?

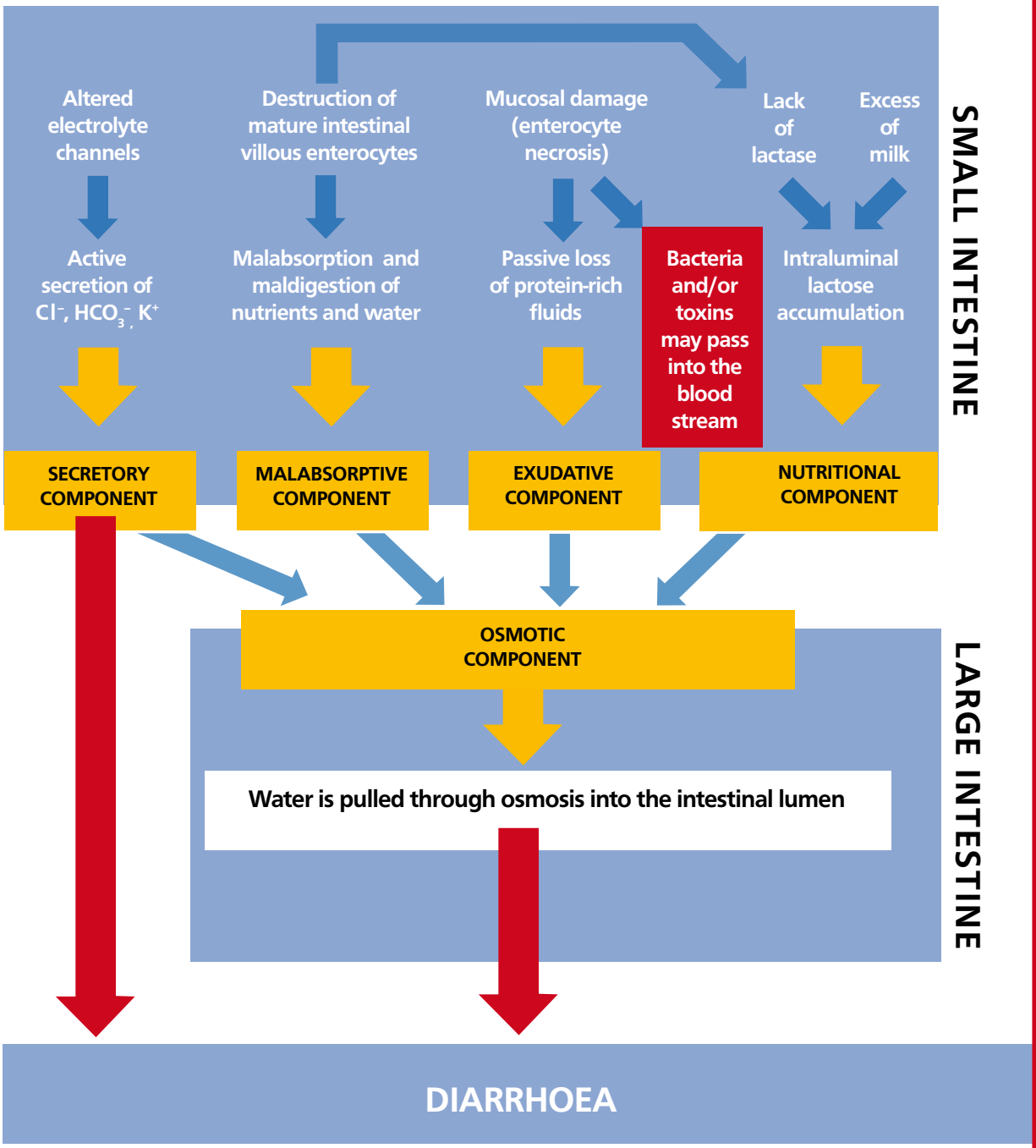
Ingestion, digestive tract secretions and extracellular fluids.

● Which mechanisms cause diarrhoea?

Malabsorption, hypersecretion, exudation and hypermotility.

b.1.2. At the enteric level

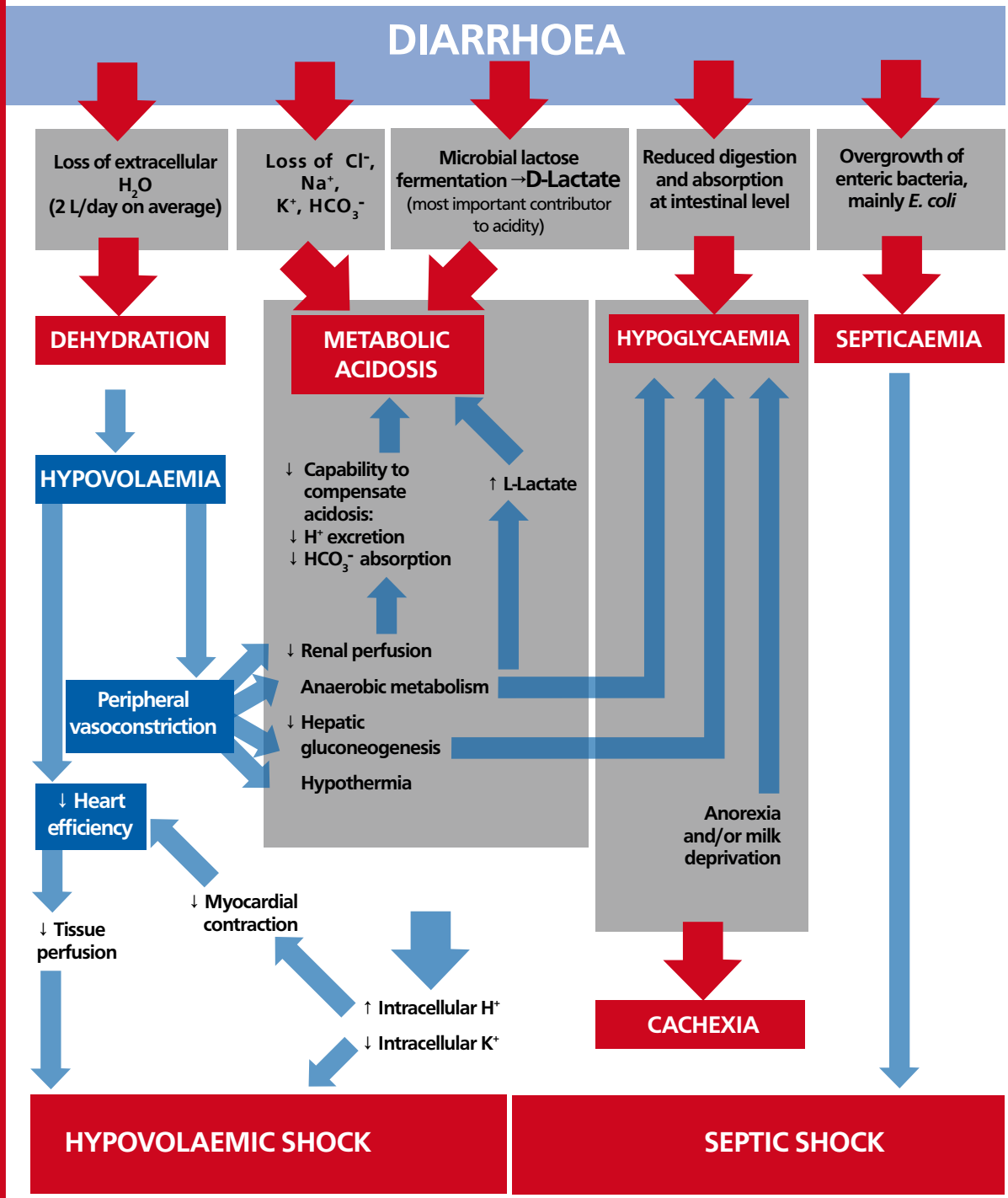
Different pathogenic causes at the enteric level may lead to some of the symptoms of calf diarrhoea.



b.1. Pathophysiology of diarrhoea

b.1.3. At the systemic level

Different pathogenic changes caused by diarrhoea will also have an effect at the systemic level.



b.2. Components of diarrhoea

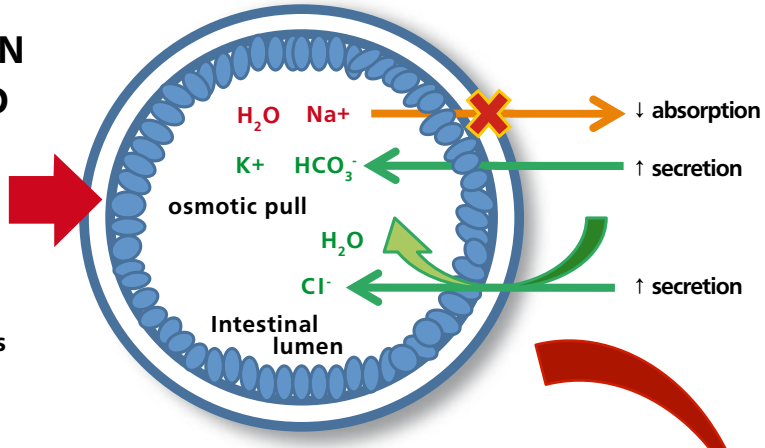
Understanding the pathophysiology of the different types of neonatal diarrhoea will help us later to better understand the pathophysiology of the different infectious and non-infectious causes of diarrhoea.

b.2.1. Secretory component of diarrhoea

This is the main mechanism leading to enterotoxigenic *E. coli* (ETEC) diarrhoea, and it also contributes to diarrhoea rotavirus and coronavirus (see page 22 - 25).

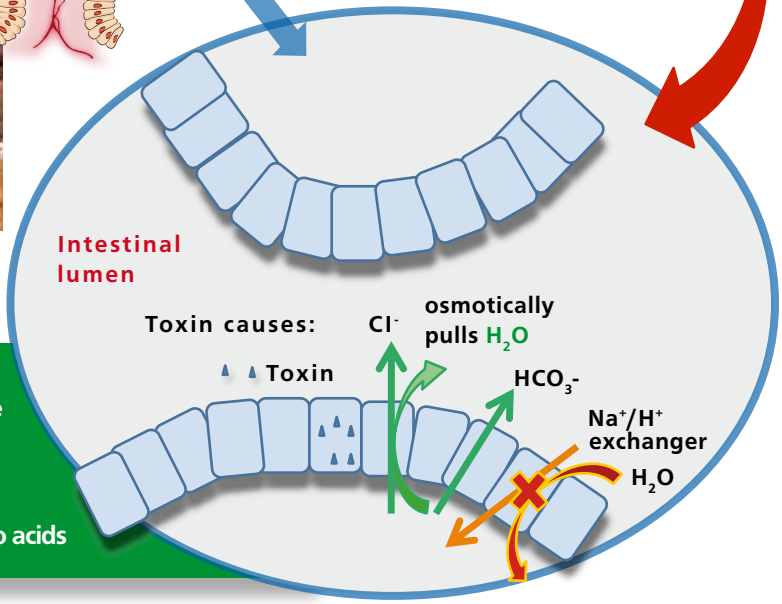
CAUSE: ALTERATION IN BALANCE OF H_2O AND ELECTROLYTES

- Little or no structural damage
- Alteration of electrolyte channels
- Increase in active secretion



Intestinal villi

Absorption is not altered, but colon's absorption ability is exceeded



TIP Since the Na^+/H^+ exchanger is not functional, we should use other routes to introduce Na^+:

- Joined with glucose
- Joined with neutral amino acids

b.2. Components of diarrhoea

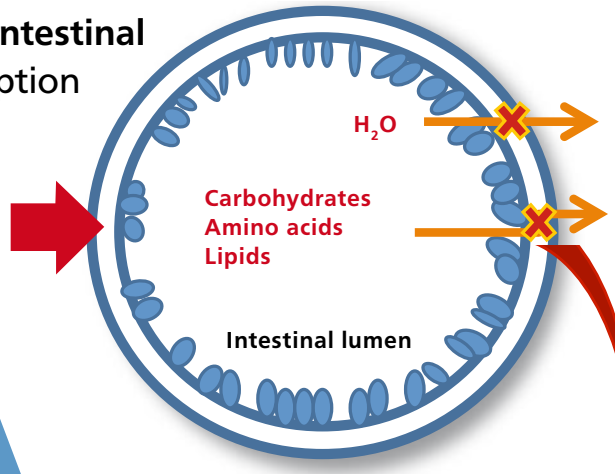
b.2.2. Malabsorptive component of diarrhoea

This mechanism is the main one involved in protozoan diarrhoea and it also contributes to diarrhoea caused by rotavirus and coronavirus (see page 24 - 25).

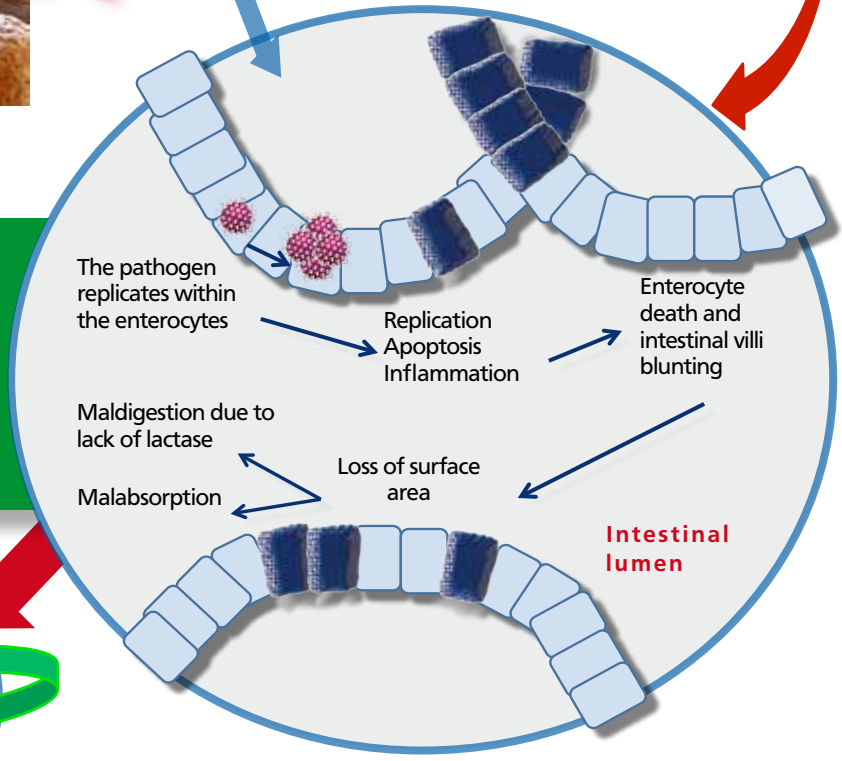
CAUSE: Destruction of mature intestinal villi enterocytes ⟶ malabsorption

In addition, it leads to **maldigestion** since lactase is produced by distal mature intestinal villi enterocytes

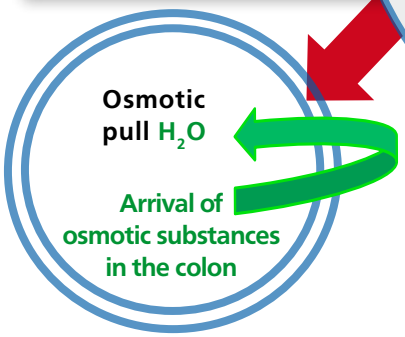
Intestinal villi



Nutrients that are osmotic substances remain in the intestinal lumen



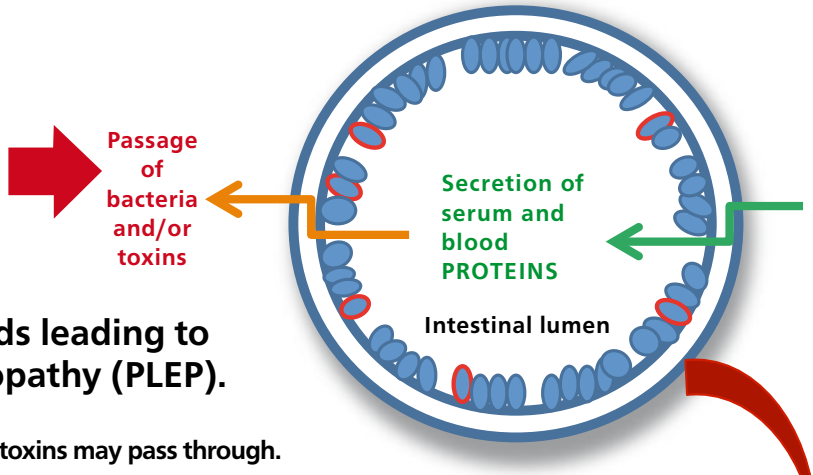
TIP Due to the lower lactase production in these diarrhoeic calves, we should administer the same total amount of milk over more meals, so that the calves receive less milk per meal.



b.2.3. Exudative component of diarrhoea

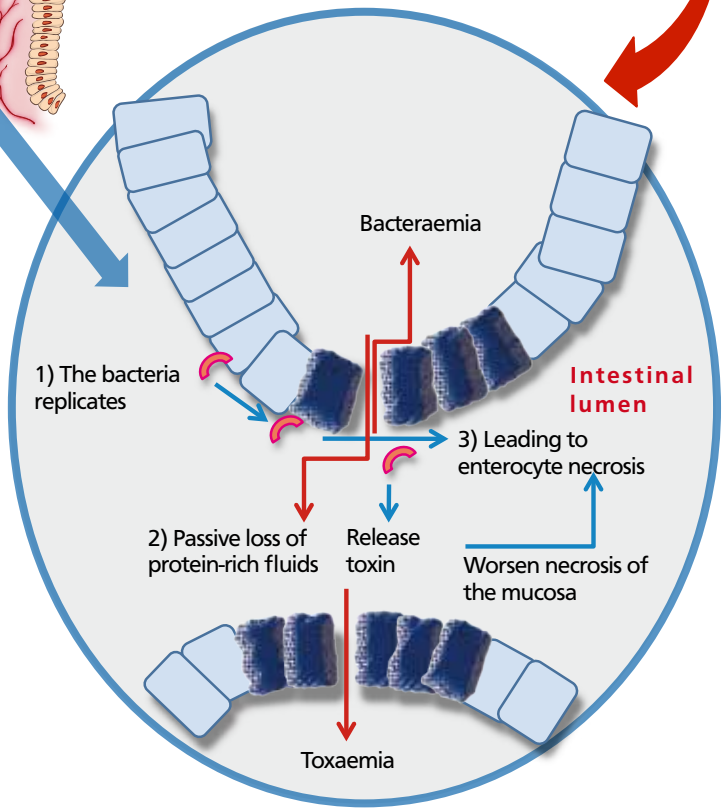
This type is the main mechanism involved in diarrhoea caused by *Salmonella spp*. and *C. perfringens* diarrhoea (see page 28 - 30).

CAUSE: Damage to the mucosal lining or brush border leads to a passive loss of protein-rich serum fluids, and a decreased ability to absorb these lost fluids leading to Protein Losing Enteropathy (PLEP).



- **In addition, bacteria and/or toxins may pass through. This leads to bacteraemia and/or toxaemia**

Intestinal villi

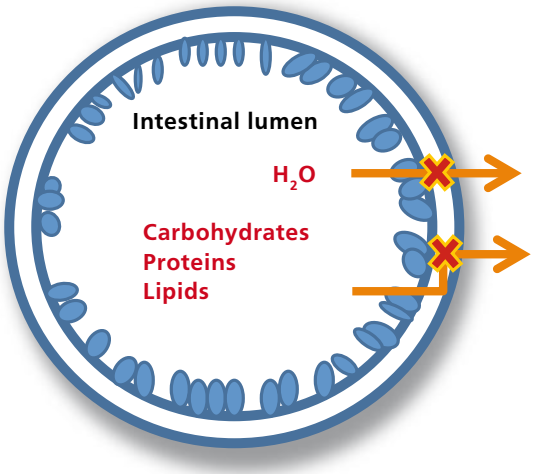


b.2. Components of diarrhoea

b.2.4. Osmotic component of diarrhoea

Due to nutritional, malabsorptive or exudative problems, lactose and other osmotic substances enter the colon, pulling water into the intestinal lumen.

In addition, the diarrhoeaic calf suffers metabolic acidosis, primarily due to microbial lactose fermentation in the intestinal lumen.



LOSS OF INTESTINAL VILLOUS ENTEROCYTES

IN COLON / CAECUM

Pass into blood stream
→ metabolic acidosis

colon lumen

Osmotically pulls H_2O

D-Lactate

Osmotic substances

Microbial hyperfermentation of carbohydrates

IN SMALL INTESTINE
Malabsorption and maldigestion

Lactose enters colon

NUTRITIONAL PROBLEMS

Excess of lactose (milk)

Lack of lactase (due to enteritis or idiopathic)

b.3. Aetiology of diarrhoea

Neonatal diarrhoea does not always have an infectious origin.
For that reason it is important to distinguish between infectious and non-infectious aetiologies.

In both cases, some causes occur more frequently ("main causes"), while others occur much less frequently ("other causes").

Types of neonatal diarrhoea based on their aetiology and frequency.

		MAIN CAUSES	OTHER CAUSES
Non-infectious	Nutritional	Rapid changes in milk quantity Mistakes in milk quantity or quality Mistakes in milk replacer management Mistakes at weaning	Heat denatured skim milk powder Allergy to non-denatured soyabean in milk replacers
	Iatrogenic dysbiosis due to different treatments, or to inappropriate use of medications (mainly oral antibiotics)		
Infectious	Bacteria	*E. coli enterotoxigenic* (ETEC) *Salmonella spp.* *C. perfringens* types A, B and C	Septicaemic *E. coli* Enteropathogenic *E. coli* (EPEC) Enterohaemorrhagic *E. coli* (EHEC) *Campylobacter jejuni* *Chlamydophila psittaci* *Yersinia enterocolitica* *Bacillus cereus* *Clostridium difficile*
	Viruses	Rotavirus Coronavirus	Calicivirus Torovirus
	Protozoa	*Cryptosporidium spp.* *Eimeria spp.* *Giardia spp.*	

b.3. Aetiology of diarrhoea

b.3.1. Non-infectious: nutritional diarrhoea

Mistakes in the management of milk replacer, in the quantity or quality of milk replacer, and in calf management are the main causes of non-infectious diarrhoea. In addition, different management failures leading to cachexia will ultimately cause diarrhoea.

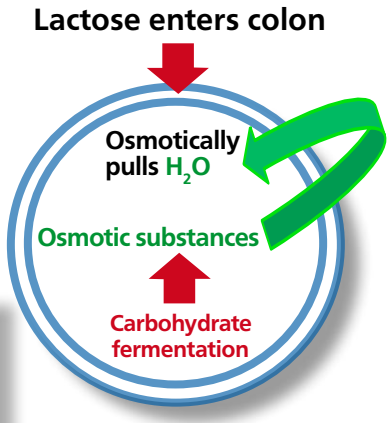
Rapid changes in composition or concentration of milk replacer

Soon after viral diarrhoea, while new enterocytes are maturing → not enough lactase

Mistake or deficit in milk replacer quality or quantity

OSMOTIC DIARRHOEA

VOLUMINOUS AND WHITISH



TIP Avoid rapid changes in composition of milk replacer, since such changes can cause acute diarrhoea without dehydration, and can predispose the animal to *E. coli* diarrhoea.

Incorrect calf management:
- Irregular feeding
- Milk fed while too cold
- Milk replacer not properly dissolved
- Drinking from an open bucket without training
- Stressors

RUMINAL DRINKING

Ruminal acidosis
Ruminitis
Ruminal hyperkeratosis
Abomasitis
Enteritis
Distended and pendulous abdomen
Absence of suction reflex
Metabolic acidosis

DIARRHOEA

WHITE CLAY DIARRHOEA

Milk deprivation in diarrhoeaic calves + malabsorption problems → even greater nutritional deficit

Weaning too soon, in the absence of proper development of ruminal papillae and intestinal villi development; or lack of adaptation to concentrated feed

Ruminal drinking can occur in calves drinking directly from open bucket without training

Competition for drinking is a stressor that can cause ruminal drinking

CACHEXIA

DIARRHOEA DUE TO HYPOPROTEINAEMIA

b.3. Aetiology of diarrhoea

b.3.2. Infectious diarrhoea

Infectious diarrhoea can be caused by different bacteria, viruses and protozoa (see table below).

Main infectious causes of neonatal diarrhoea in calves

		Age	Transmission	Type of diarrhoea
Bacteria	*E. coli* (ETEC)	1 - 5 days	Faecal-oral	Secretory
	Salmonella spp.	4 - 28 days and older	Faecal-oral 60% and through mucosas	Exudative
	C. perfringens types A, B and C	1 - 15 days and older	Faecal-oral	Exudative
Viruses	Rotavirus	5 - 14 days	Faecal-oral	Malabsorptive-osmotic and secretory
	Coronavirus	5 - 30 days	Faecal-oral and nasal secretions	Malabsorptive-osmotic and secretory
Protozoa	*Cryptosporidium parvum*	5 - 20 days	Faecal-oral (faecal oocysts already infective)	Malabsorptive-osmotic
	Eimeria spp.	> 3 weeks	Faecal-oral (faecal oocysts need to sporulate to became infective)	Malabsorptive-osmotic
	Giardia spp.	> 15 days	Faecal-oral	Malabsorptive-osmotic

b.3.2.1. *Escherichia coli*

E. coli **is a Gram-negative member of the family Enterobacteriaceae, which is part of the normal intestinal commensal flora of most animals.**

Most strains are harmless and only some strains are capable of causing disease.

To induce disease, the strain requires some virulence factors, which are classified by distinctive antigens. They are divided into groups based on pathogenic mechanisms as seen in the table below.

Classification of pathogenic strains based on pathogenic mechanisms

Strain	Subtype/ Group/ Antigen	Mechanism	Results
Enterotoxigenic E. coli (ETEC)	90% F5 antigen (K99)	Heat-stable toxin (STa)	Calves younger than 1 week: secretory diarrhoea
	Lower frequency FY-F41	Lower fimbrial receptors	Calves older than 1 week: asymptomatic carrier
Enteropathogenic E. coli (EPEC)	Verotoxigenic	Microvilli disruption	Mainly calves aged 1-5 weeks; infects large intestine and may cause malabsorptive diarrhoea
	Attaching and effacing (AEEC)		
Septicaemic (invasive)	O78, O137, O153, CS31A	When the calf receives a low level of colostrum antibodies and/or a very high bacteria pressure	Septicaemia
Apathogenic or very low pathogenicity	Enterohaemorrhagic *E. coli* (EHEC) *(Shigatoxin E. coli)* Includes O157:H7	Without animal health implications, but cattle are the main reservoir: significant public health implications	

DO NOT CONFUSE...

Since *E. coli* belongs to the normal intestinal flora, finding it in faecal cultures has no diagnostic value without evidence of pathogenic virulence factors.

Enterotoxigenic *E. coli* (ETEC)

ETEC is the most relevant strain involved in neonatal diarrhoea

Two major attributes make it virulent: the ability to colonise the intestine and the capacity to produce toxins that stimulate the secretion of electrolytes and water by intestinal mucosa.

ETEC key factors

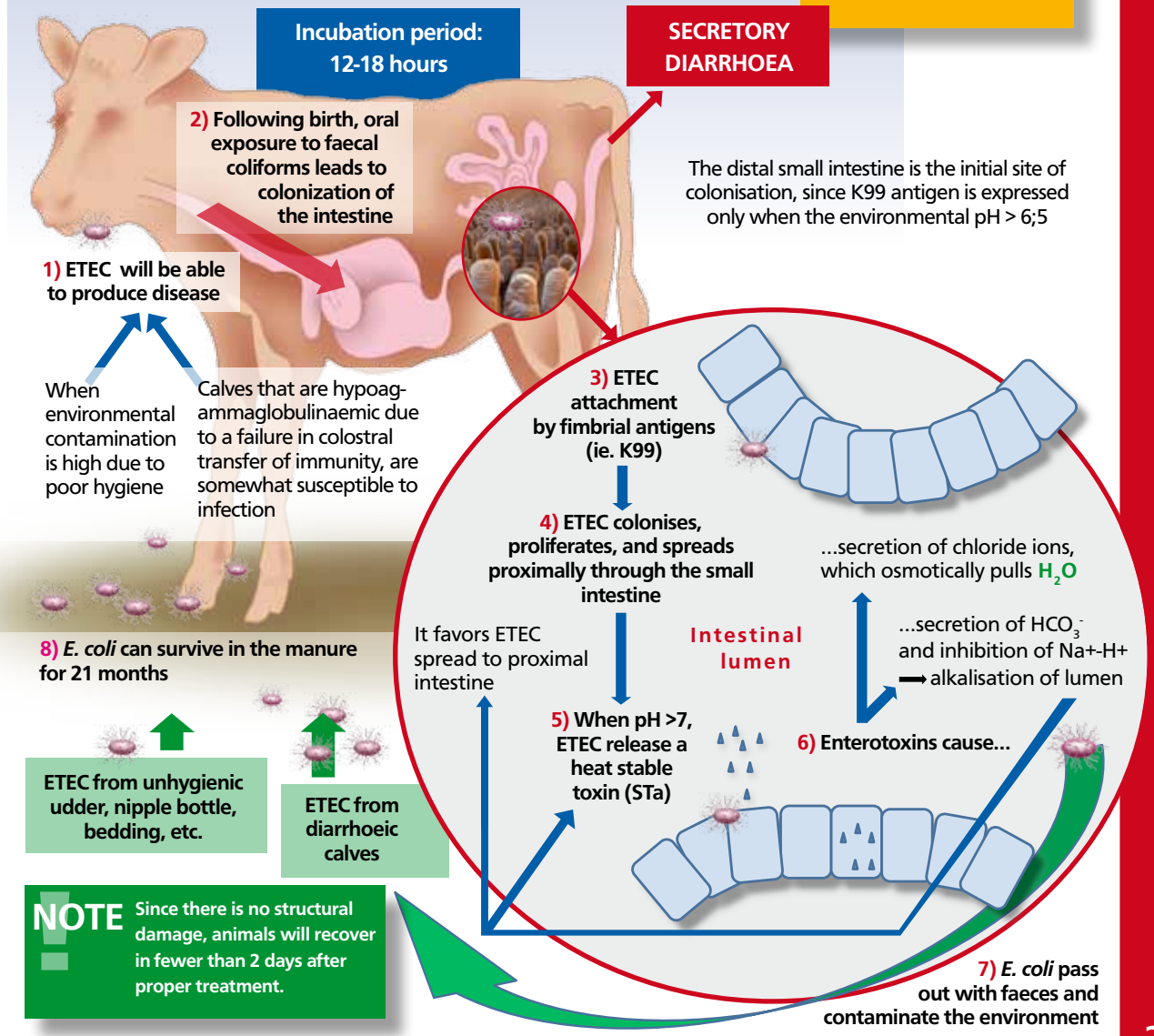
First: It must survive abomasum pH

Second: K99 attachment to ileum and heat stable toxin (ST) expression, which depend on intestinal pH level (>6;5 for K99 and >7 for ST).

CAUTION...

ETEC only affects calves within the first 5 days of life when abomasal pH is 6-7, before it descends to < 2.

Be aware of this when using alkalising oral electrolytes for oral rehydration!!



NOTE Since there is no structural damage, animals will recover in fewer than 2 days after proper treatment.

b.3.2.2. Rotavirus and Coronavirus

	ROTAVIRUS	CORONAVIRUS
Characteristics	Ubiquitous. Commensal in bovine animals, 90-100% are seropositive. Seven serogroups recognized (A-G). In neonatal diarrhoea serogroup A is the most commonly reported.	Less frequent than rotavirus. May also be involved in cow winter dysentery and respiratory disease complex.
Shedding	In diarrhoeaic calves only 40% shed the virus in their faeces due to previous shedding of cells harbouring the virus. Healthy calves can also shed the virus (18%) without developing disease [4].	In diarrhoeaic calves, 8-69% shed the virus in their faeces due to previous shedding of cells harbouring the virus. Some healthy calves (0-24%) shed the virus without developing disease [21], and 70% of clinically normal cows shed intermittently at low levels.
Location of pathogenic effect	Small intestinal area (confined mainly to the duodenum).	Extensive intestinal areas, beginning in proximal small intestine and usually spreading throughout the jejunum, ileum, and colon.
Lesions	Less severe and lower mortality. Death is generally associated with mixed infections.	More severe and higher mortality. Clinical signs last longer, as infection leads to destruction of crypt enterocytes and colonocytes.

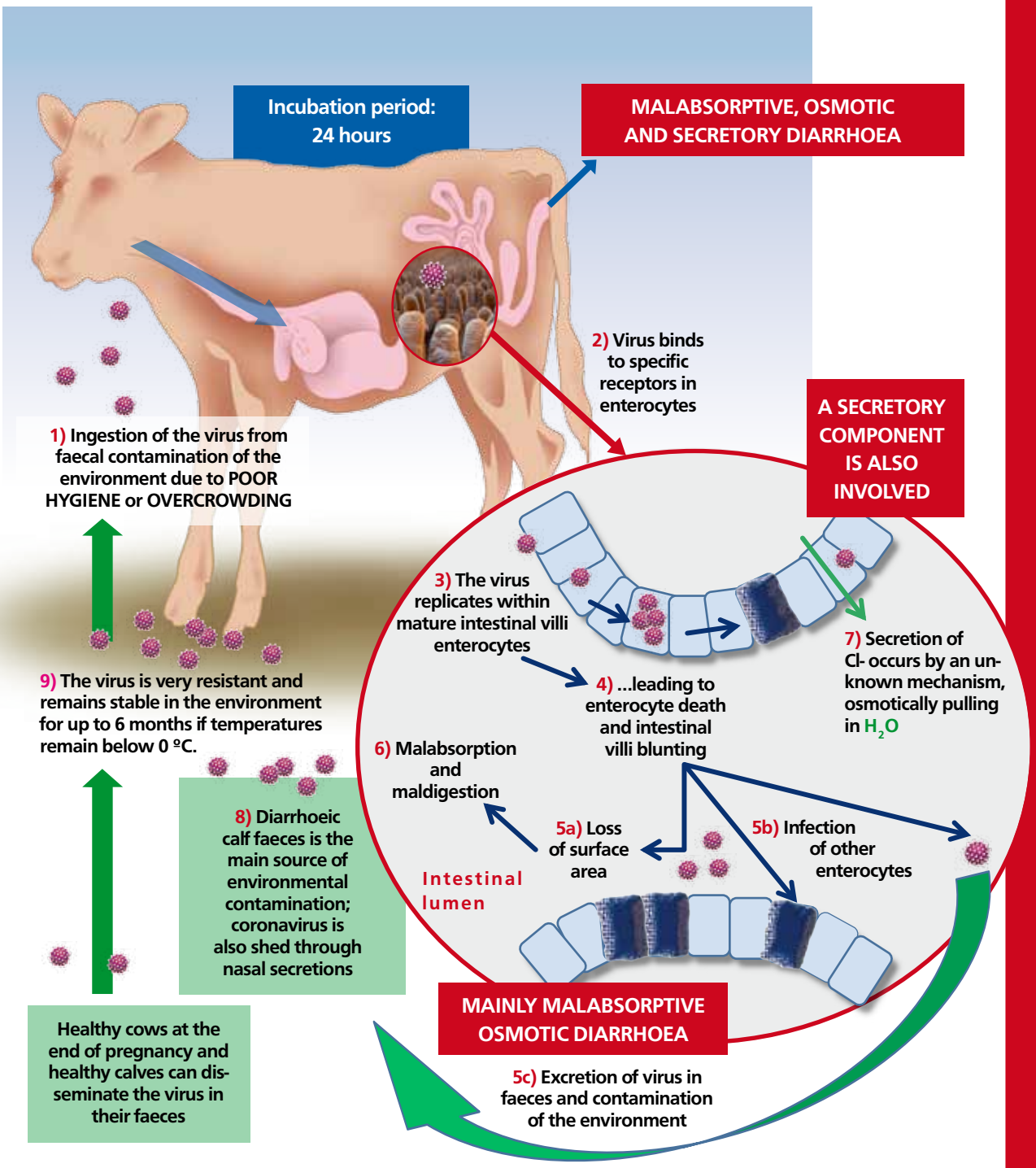
CAUTION

As the viruses are cytolytic, some animals with viral diarrhoea can be negative for viruses, because the cells harbouring the virus have been previously shed through faeces. Some calves without disease shed the virus in their faeces.

NOTE

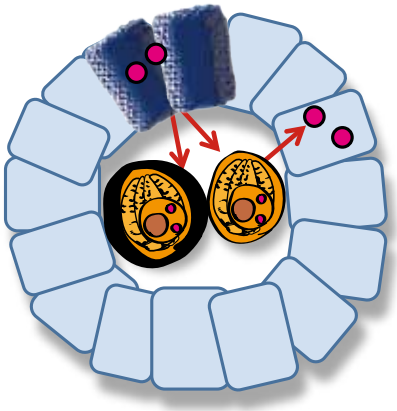
Since the virus causes structural damage, animals take longer to recover than from *E. coli* (2-4 days) after proper treatment

Pathogenic mechanism Rotavirus and Coronavirus (similar for both)



b.3.2.3. *Cryptosporidium spp.*

- Mainly caused by *Cryptosporidium parvum*, a protozoan that affects 170 species including bovine species. Occasionally *C. andersoni* is described in beef calves.
- *C. parvum* is zoonotic. However, severe outbreaks are rare. The majority of human cases are small foci of infection, which may be caused both by *C. parvum* and *C. hominis* [22].
- Depending on the housing system, density and management of the calves up to 100% of calves that become infected shed oocysts in their faeces.
- *C. parvum* affects calves older than 4 days, usually those between 10-20 days of life.



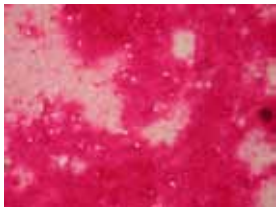
Thin-walled oocysts allow pathogen to infect other enterocytes in the same calf (autoinfection)

Thick-walled oocysts pass out with faeces and contaminate the environment

NOTE

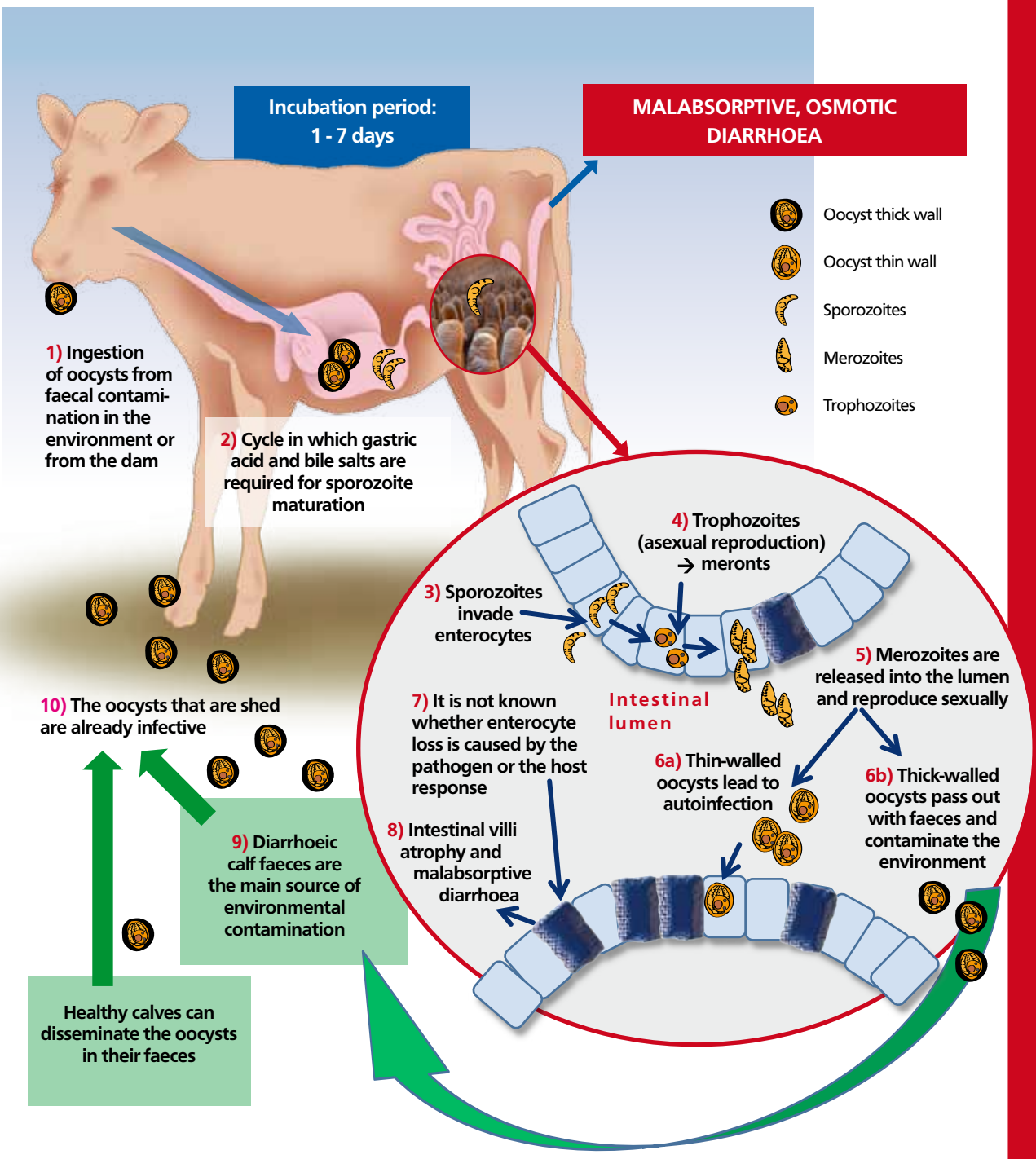
Because of auto-infection, a very low dose of pathogen can induce disease.

- Thick-walled oocyst are very resistant to most disinfectants, adhere very well to plastic materials (such as nipples bottles, buckets, etc.), and can remain viable for about 18 months in cool, damp or wet environments. Oocyst infectivity can be destroyed by 10% ammonia, formalin or exposure to desiccation and temperatures above 60° C.
- Concequences of infection in the calf:
  Usually remains subclinical or chronic.
  Sometimes calves develop a bloody, watery diarrhoea ⟹ dehydration and electrolyte loss.
  Fatal cases are usually associated with mixed infections involving other pathogens as well.
- In immunocompetent calves, infection is self-limiting and may clinically resolve spontaneously leading in most cases to lower average daily weight gains, and in some rare cases even to non economic value
- Calves appear to be resistant to subsequent infection after an initial episode of *C. parvum* diarrhoea



Modified Ziehl-Neelsen staining of faeces showing *Cryptosporidium spp.* as white refracting spheres

Crysptosporidium parvum



b.3.2.4. *Salmonella spp.*

- Gram-negative member of the Enterobacteriaceae family. The serotypes most frequently involved in diarrhoea: *S. enterica* Dublin, which affects calves but also adult cattle; and *S. enterica* Typhimurium, which mainly affects calves younger than 2 months old.
- Zoonotic agent
- Epidemiology
  - Sporadic cause of neonatal diarrhoea
  - Epizootic in feedlots and rearing units due to mixing of calves from multiple farms
  - Endemic infection associated with fascioliasis has been described in some dairy herds.

CAUTION...

Carriers can remain asymptomatic for years, developing the disease or shedding the bacteria when the animal is immunosuppressed.

- Affects calves older than 4 days (usually 4-7 weeks) and diarrhoea is not always present
- Especially common in stressed calves, such as when they are weaned onto milk replacers
- Healthy calves have not been reported to shed *Salmonella spp.* in their faeces.
- May be associated with terminal dry gangrene as a consequence of septicaemia (calves) or abortion (serotypes Typhimurium and Dublin)
- Sources of infection in the herd
  - Sporadic causes of neonatal diarrhoea
  - Contaminated feed / equipment
  - Faeces of birds (pigeons and gulls) or wild mammals
  - Use of bird faeces as pasture fertiliser.
- Transmission routes
  - Faecal-oral from diarrhoeaic and asymptomatic carriers (MAIN ROUTE)
  - Oronasal secretions and urine before onset of symptoms
- Infection can become systemic: septicaemia, peritonitis, pneumonia, etc.

b.3.2.5. *Clostridiun perfringens*

- Gram-positive anaerobic, spore-forming (environmentally-resistant) bacterium of the genus *Clostridium*. Clostridia are ubiquitous. Neonatal calves are affected by types A, B and C. Type D, responsible for classic enterotoxaemia, does not affect neonatal animals.

- Their pathogenicity is based on the production of toxins. Toxin-α is the most important for *C. perfringens* type A, while toxin-β is the most important for *C. perfringens* types B and C. Trypsin and low pH inhibit the effect of toxin-β.

  - *C. perfringens* types B and C affect neonatal calves from the first day of life (due to colostrum antitrypsin) or older calves (due to overfeeding and the ensuing deficit of trypsin).
    The bacteria cause haemorrhagic enteritis in more or less extensive areas, depending on whether type B or C predominate, respectively.

  - *C. perfringens* type A affects calves mainly during days 0-14 of life, producing haemorrhagic abomasitis and enteritis.

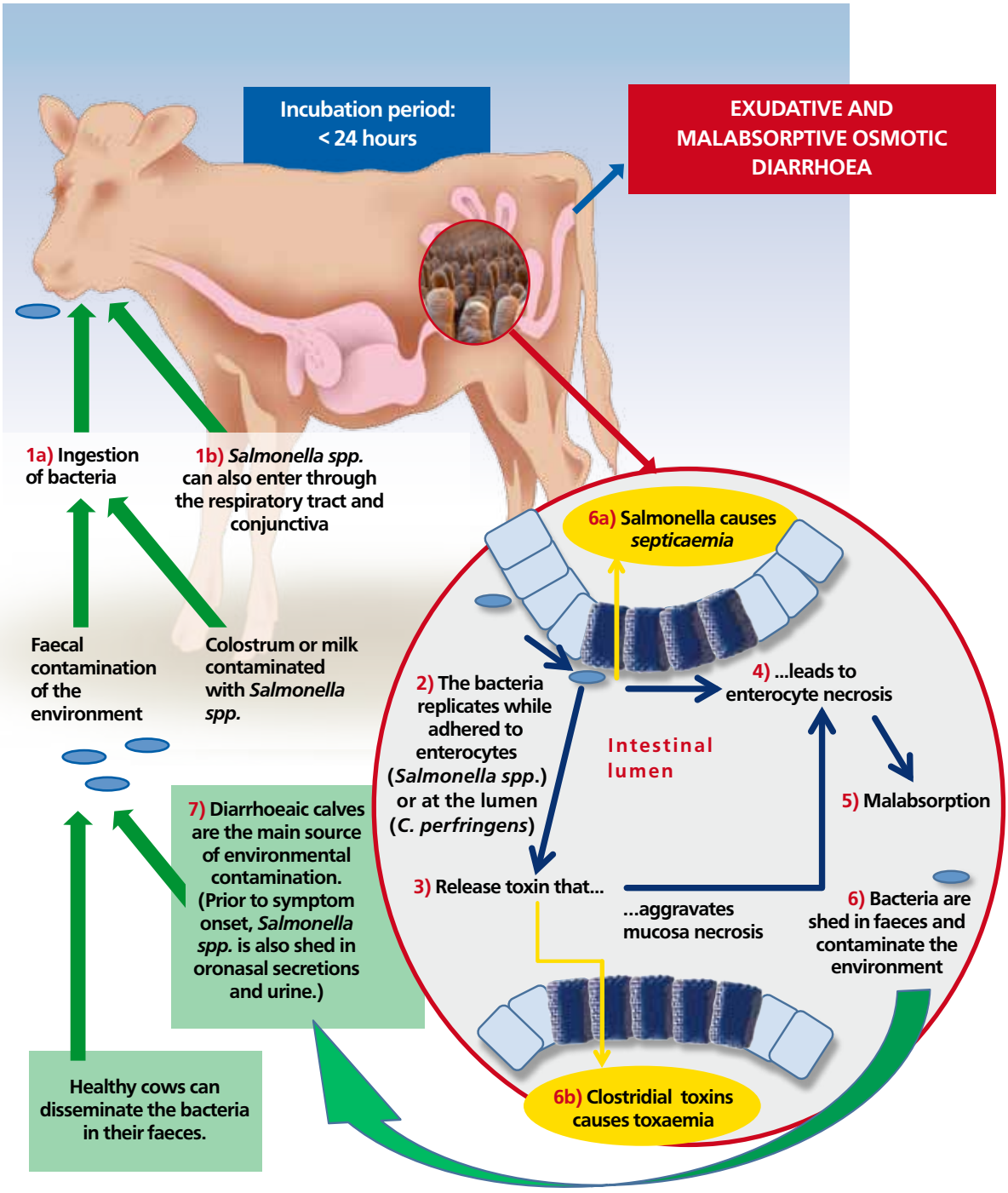
NOTE

Trypsin and low pH inhibit the effects of toxin-β.

Colostrum antitrypsin and high abomasum pH allow toxin-β to exert its effects in neonatal calves.

Cases and outbreaks after abrupt changes in the amount or quality of feed ⟶ bacteria proliferate because there is not enough trypsin

Pathogenic mechanism *Salmonella spp.* and *C. perfringens* (similar for both)

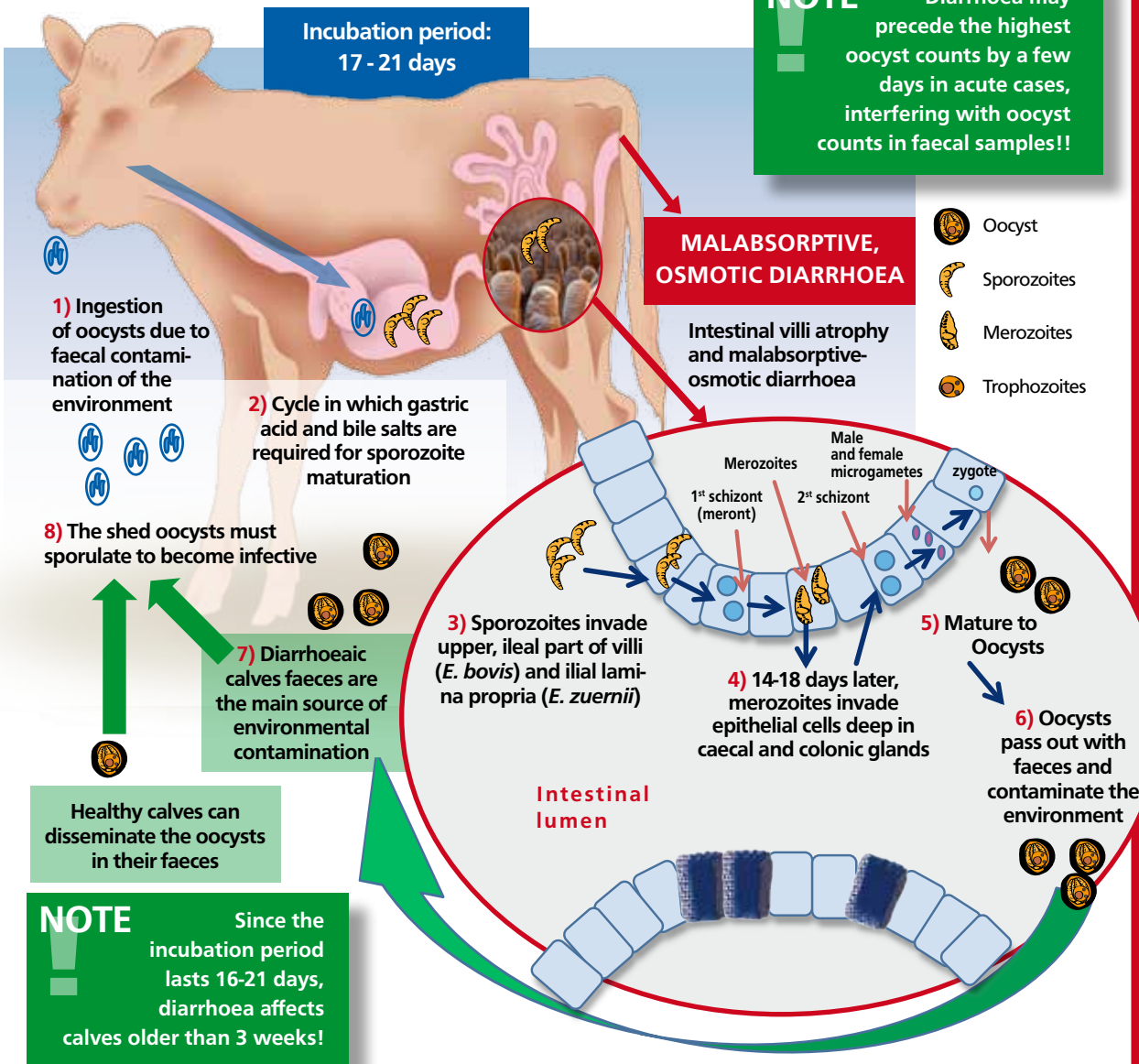


b.3.2.6. Eimeria spp.

- Coccidiosis is an infection produced by protozoa with a high host specificity. Although there are more than a dozen species, only *Eimeria bovis*, *Eimeria zuernii,* and *Eimeria alabamensis* are responsible for severe clinical disease.
- Although infection can occur during the first days of life, the incubation period is 17 days and as a result, diarrhoea appears in calves older than 3 weeks, most frequently among calves aged 2 - 6 months.
- Adult cattle can act as asymptomatic carriers.
- Although the pathogenesis of neurological signs of coccidiosis is unknown, their presence indicates a poor prognosis.

Sporulated *Eimeria* oocyst

NOTE Diarrhoea may precede the highest oocyst counts by a few days in acute cases, interfering with oocyst counts in faecal samples!!

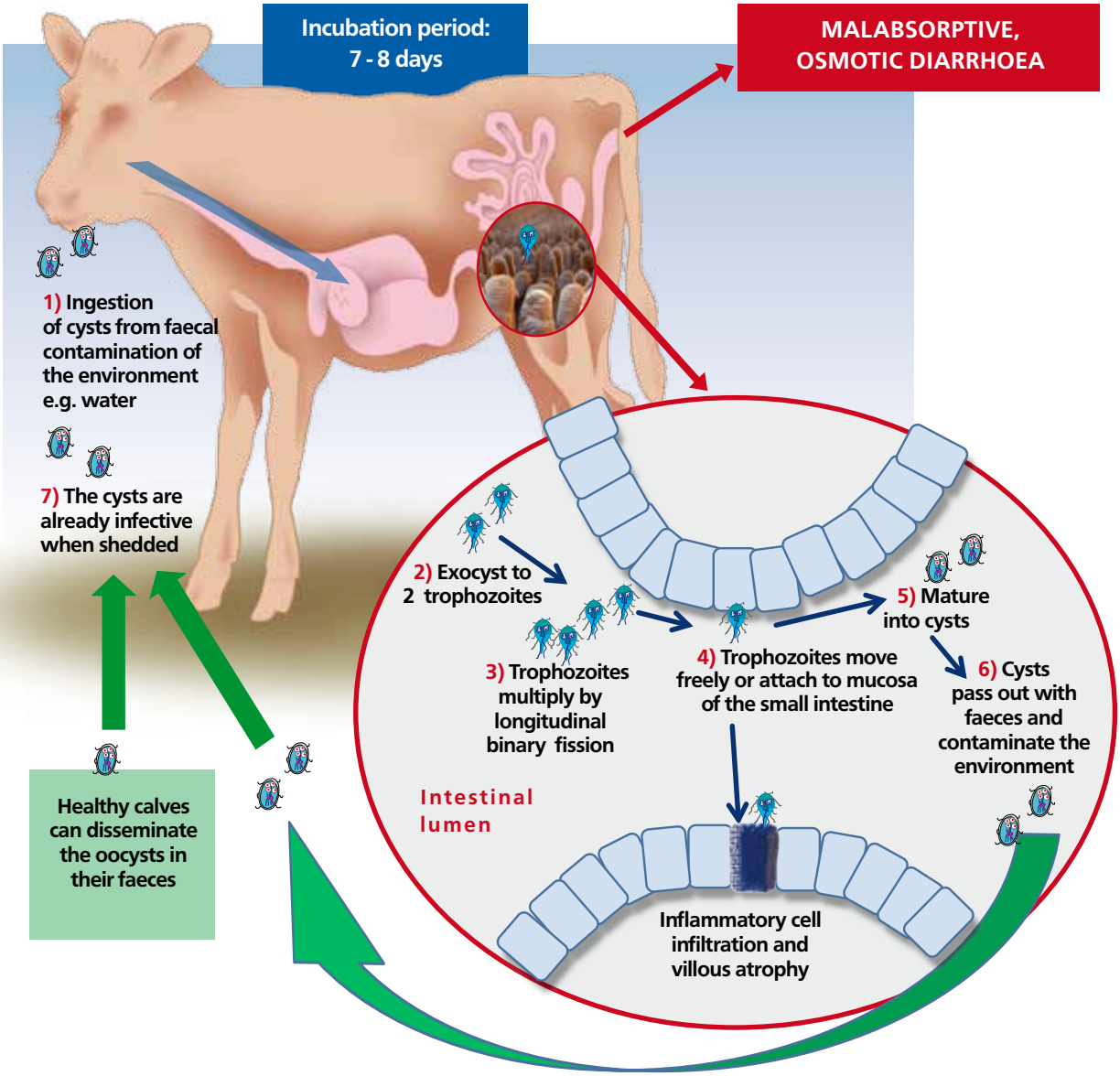


NOTE Since the incubation period lasts 16-21 days, diarrhoea affects calves older than 3 weeks!

Unknown whether enterocyte loss is caused by the pathogen or the host response.

b.3.2.7. Giardia spp.

- Giardiasis is caused by *G. duodenalis* (previously known as *G. lamblia* and *G. intestinalis*), an anaerobic flagellated protozoan found in up to 60% of diarrhoeaic calves.
- Althought asymptomatic infection can be found, Giardia may cause disease in calves under some circumstances, and retardation of growth is frequently observed.
- It may cause diarrhoea in calves older than 15 days.
- Although *G. duodenalis* causes diarrhoea in people, evidence suggests that cattle and people can be infected by identical subtypes (e.g. Assemblage A) [22].



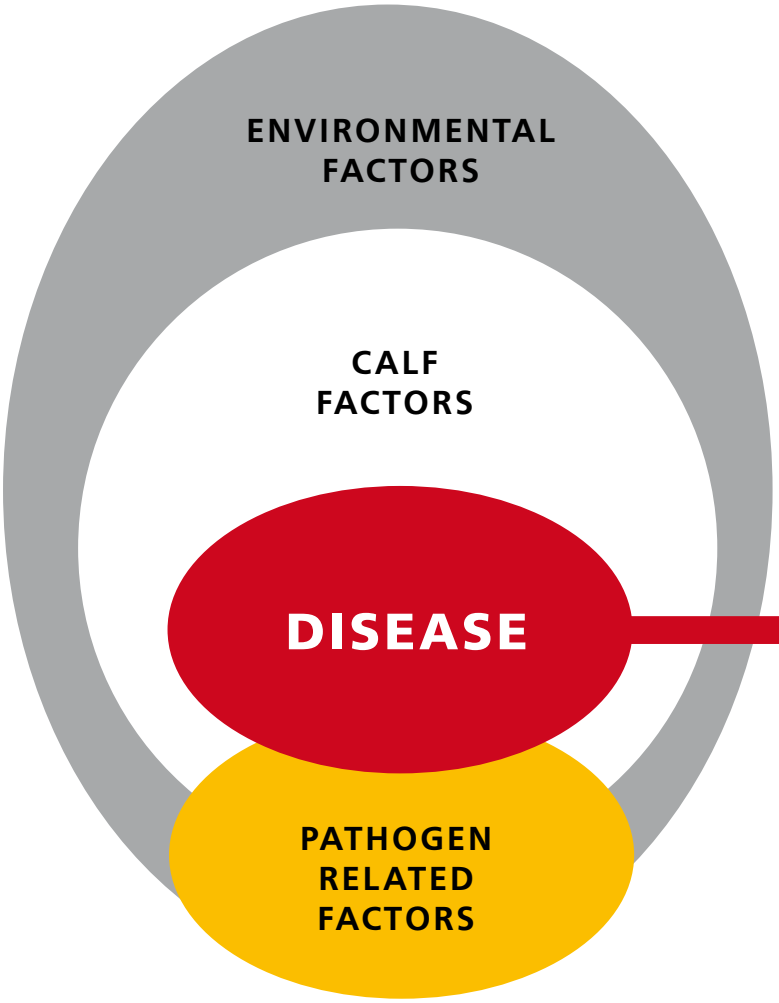
b.3.2.8. Other enteropathogens

- **BACTERIA**
  - ***Campylobacter jejuni:*** its importance is limited to its zoonotic potential.
  - ***Chlamydophila psittaci, Yersinia enterocolitica*** and ***Bacillus cereus*** are considered to cause sporadic cases.
  - ***Clostridium difficile*** is probably an emerging problem.

- **VIRUSES**
  - ***Calicivirus*** and ***torovirus*** have been implicated in neonatal diarrhoea, but their pathogenicity is low or unproven and their prevalence is sporadic.

b.4. Neonatal diarrhoea risk factors

The causes of diarrhoea are complex and usually involve primarily an interaction among the enteropathogen, the calf and environmental factors.



Although enteropathogens are often present (most are ubiquitous and are shed by healthy animals), diarrhoea is not always present.

Their presence is necessary but not sufficient to develop the disease.

The calf will become diarrhoeaic only when additional environmental and/or animal risk factors are present.

Hindquarter of a calf with diarrhoea smudged with faeces

Thus, the disease is the sum of many factors ⟶ diarrhoea is a multifactorial disease

REMEMBER...

There are farms where environmental factors are present without any neonatal diarrhoea problem. However, when a diarrhoea problem occurs ⟶ correcting those factors will be essential for resolving the diarrhoea problem

b.4. Neonatal diarrhoea risk factors

b.4.1. Environmental factors

a. Herd characteristics and management practices

DECREASE

- Individual maternity pen: individual, clean and disinfected between calvings

- Administration of colostrum with nipple bottle or feed tube

- Calves housing: individual pens without direct contact

- Pens are disinfected, and moved between successive uses

INCREASE

- Sucking of colostrum directly from the dam

- Housing calves in groups or direct contact among calves

- Commingled age groups comprising peripartum cows, sick cows or recently weaned calves

- Leaving the calf longer than 1h with the dam

- Mild replacers (perhaps due to inferior quality), or irregular feeding practices ⟶ dietetic diarrhoea and higher incidence of enteric colibacillosis

- Larger herd size

b. Level of environmental hygiene

Poor hygienic practices and overcrowding increase the risk of infection and the risk of diarrhoea.

Non-hygienic bedding in the maternity pen

Overcrowding in individual pens

c. Weather influences

In the case of beef cattle, confining calving cows during inclement weather is a common practice, and overcrowding may be followed by an outbreak of calf diarrhoea.
In the case of dairy cattle, many veterinarians have observed a relationship between adverse climatic conditions and colibacillosis in calves, but few epidemiological data support the claim.

During dry and hot weather, the risk of diarrhoea appears to be lower

Colibacillosis has been linked with inclement weather

b.4. Neonatal diarrhoea risk factors

b.4.2. Calf factors

a. Weak calf syndrome

Lack of vigor of the calf at birth due to *intrapartum* hypoxia and acidosis from a difficult birth or dystocia, which implies a lower and/or later intake of colostrum. For that reason the calf will not reach the necessary level of serum colostral antibodies.
Signs of dystocia: depression and acidosis, scleral petechia, and delayed postnatal behaviour.

Calf depressed, unable to stand and suckle colostrum on time

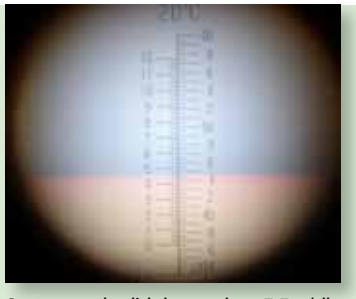
Scleral petechia due to acidosis

Edematous head which hinders calf suckling

Delayed adoption of sternal recumbency

b. Calf immune status

The specific immunity of the newborn calf is dependent on colostrum intake. The lack of this passive transfer of immunity is the MAIN RISK FACTOR for neonatal diarrhoea.



Serum total solids lower than 5.5 g/dl in a 3-day-old calf, indicating a possible failure of passive transfer of immunity

TIP **Dam vaccination increases specific antibodies against enteropathogens in colostrum, which can enhance calf immune status when immune transfer occurs at the appropriate time and in adequate quantities**

c. Age

Calf age determines the onset of the different pathogens involved in neonatal diarrhoea
(see page 49).

Diarrhoea in a calf younger than 5 days is usually due to ETEC

Diarrhoea in a 30-day-old, cachectic calf with hypoproteinaemia

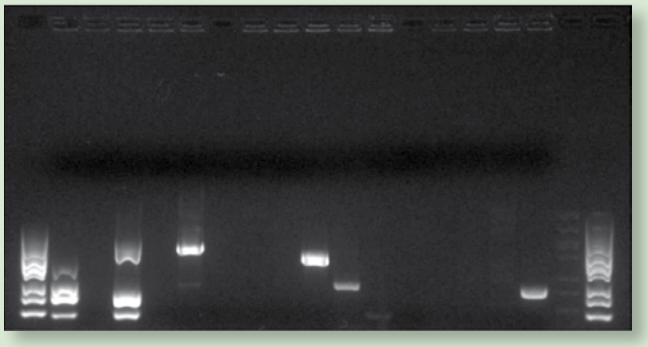
b.4. Neonatal diarrhoea risk factors

b.4.3. Pathogen-related factors

Pathogenicity

Differences in virulence among serotypes determine whether they are capable of inducing disease and the severity of that disease.

For example, *E. coli* has only some pathogenic strains. Enterotoxigenic *E. coli* (ETEC) is the most important one involved in neonatal diarrhoea; nevertheless, there are others involved in others diseases such as septicaemic *E. coli* or enterohaemorrhagic *E. coli* (EHEC).



31.1. Detection of pathogenicity factors in *E. coli* by multiplex PCR

Environment-pathogen-host interface

Ubiquity.
Some pathogens are ubiqitous and very prevalent in farms such as *E. coli, rotavirus, coronavirus,* and *C. parvum*. Other pathogens may be introduced on to the farm, such as *Salmonella spp.*

Concurrent infection with different pathogens. This increases the risk of diarrhoea and its severity, and it extends the age of susceptibility.

High bacterial load leads to high infectious pressure

b.4. Neonatal diarrhoea risk factors

b.4.3.1. Ubiquity

Most enteropathogens are shed not only by diarrhoeaic calves but also by healthy calves and cows

MAIN SOURCE OF CONTAMINATION

Calves with diarrhoea

1 g of diarrhoeaic faeces may contain:

10^{10} enterotoxigenic *E. coli*
10^{10} viral particles
10^{7} *C. parvum* oocysts
10^{9} *Salmonella spp.*

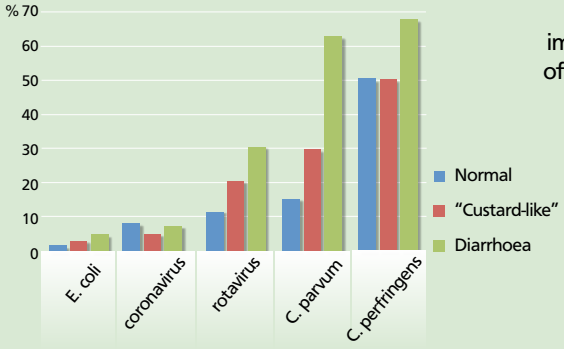
RESERVOIR OF INFECTION

Healthy Calves

Case control studies indicate that enteropathogens commonly found in diarrhoeic calves can also be present in healthy calves, but at a lower prevalence

Cows

Primary source of neonate infections with *Salmonella spp.*, *E. coli,* rotavirus...



Apparent animal prevalence of enteropathogens stratified by three categories of faecal consistency in Dutch dairy calves (aged 1–21 days) [23]

Postpartum immunosuppression of the dam increases fecal shedding of enteropathogens at parturition

- **FAECAL-ORAL TRANSMISSION IS THE MAIN ROUTE OF INFECTION** for most enteropathogens.
  They spread within a herd through the faeces of infected animals and all the inanimate objects that can be contaminated by them:
  - Bedding and dirty calf pens
  - Nipple bottles and buckets
  - Boots, tools, clothing
  - Feed and water supplies

- Some enteropathogens have additional transmission routes:
  - Airborne or via nasal secretions: the mucosa of the upper respiratory tract and conjunctiva (e.g. *Salmonella spp.)*
  - Navel

CAUTION...

Due to the ubiquity of most enteropathogens, faecal shedding profiles can be validated as causative agents when they are consistent with epidemiology (mainly age of onset) and post-mortem microscopic lesions.

b.4. Neonatal diarrhoea risk factors

b.4.3.2. Concurrent infection

MIXED INFECTIONS ARE.... MORE FREQUENT THAN SINGLE ONES

Diarrhoea associated with rotavirus, coronavirus or *E. coli* must be considered in the context of "acute undifferentiated diarrhoea of newborn calves" because mixed infections are more common than single ones [24].
Mixed rotavirus and coronavirus may occur in the same diarrhoeaic calf in the presence or absence of *E. coli.* In addition, *Cryptosporidium spp*. is frequently associated with rotavirus and/or *E. coli*, and in some cases, when the calf is close to recovery, it can suffer a successive coccidial infection.

The most common mixed infection is rotavirus- *Cryptosporidium spp*., which is responsible for 10% of cases of neonatal diarrhoea between 2 and 7 days of age, and for 55.5% of cases between 8 and 14 days [25].

MIXED INFECTIONS ARE.... MORE SEVERE THAN SINGLE ONES

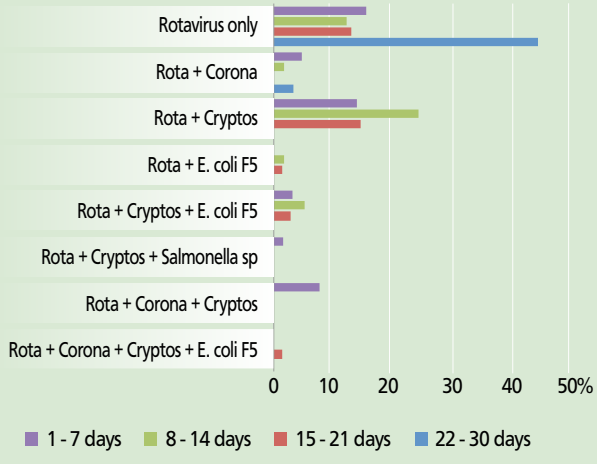
Pathogenicity and symptomatology will be the sum of the effect of the different pathogens involved, which may sometimes reinforce themselves (synergism), aggravating the clinical picture and increasing the morbidity and mortality.

MIXED INFECTIONS.... EXTEND THE AGE OF SUSCEPTIBILITY

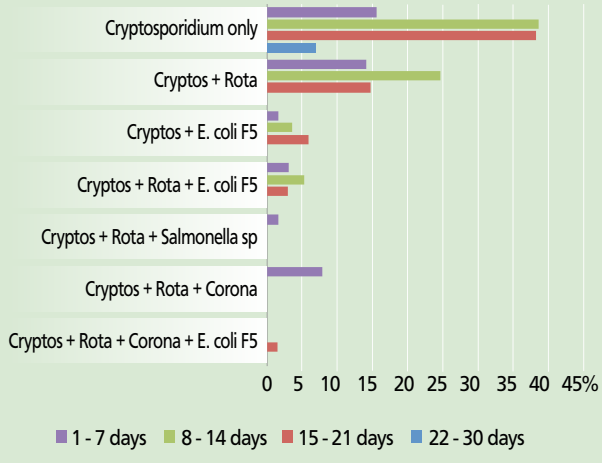
Enterotoxigenic *E. coli (ETEC)* infection in calves older than 4-5 days will in most cases be associated with concurrent rotavirus infection.

TIP

Dam vaccination will reduce severity of diarrhoea, including diarrhoea caused by *Crypstosporidium*, because it prevents concurrent infections.



Detection of rotavirus and its combination with other enteropathogens in different age groups of diarrhoeaic calves [26]



Cryptosporidium and concurrent infections with other enteropathogens in different age groups of diarrhoeaic calves [27]

c.1.Investigation of the diarrhoeaic calf
c.1.1. Identifying the disease
c.1.1.1. Clinical signs

When we are faced with a calf with diarrhoea, we should evaluate the score of diarrhoea, dehydration, and the presence of acidosis and/or septicaemia in order to establish a correct diagnosis, prognosis and treatment.

Parameters that should always be evaluated

Diarrhoea score

Septicaemia

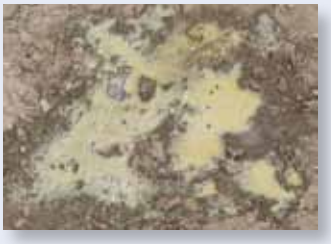
Dehydration score

Acidosis

c.1. Investigation of the diarrhoeaic calf
c.1.1. Identifying the disease
c.1.1.1. Clinical signs

Diarrhoea score

Scoring system according to McGuirk [28] modified

Score	Characteristics		
0	Normal faeces, with the consistency of pudding. Meconium (first faeces of the calf).		 Meconium
1	Semiformed or pasty, similar to yogurt.		
2	Loose but enough consistency to remain on bedding. Consistency of syrup.		
3	Watery faeces that drop through bedding material.		
B	Blood is present.	 B	 BM
BM	Blood and mucus present.		

c.1.Investigation of the diarrhoeaic calf
c.1.1. Identifying the disease
c.1.1.1. Clinical signs

Dehydration score

To evaluate dehydration use the following parameters:

- Enophthalmos
- Skin tenting: to evaluate it, pinch a fold of skin and count the seconds that it takes to flatten
- Gum color and moisture: normal gums should be pink and damp
- Attitude during milk feeding: suckling reflex should be strong in healthy calves.
- Temperature of extremities.

Absence of enophthalmos in a calf

TIP

When dehydration reaches 8% intravenous fluid administration is recommended!

Evaluation of degree of dehydration in diarrhoeic calves based on clinical symptoms.

Degree of dehydration	Symptoms			
	Enophtalmos	Skin tenting	Gums	Extremities
0%	Absence	less than 2 seconds	wet	warm
2%	1 mm	2 seconds	dry	warm
4%	2 mm	4 seconds	dry	warm
6%	lightly sunken eyes (3 mm)	5 seconds	dry	warm
8%	sunken eyes (4 mm)	6 seconds	dry	cool
10%	very sunken eyes (6 mm)	7 seconds	dry	cool
12%	very sunken eyes (7 mm)	>8 seconds	pale	cool
>14%	>8 mm	>10 seconds	pale	cool

Sunken eyes in 4-mm enophthalmos in a calf

Very sunken eyes in 7-mm enophthalmos in a calf

Skin tenting in a calf

DO NOT CONFUSE... **Dehydration with enophthalmos due to reduction of fat stores in a cachectic calf. To differentiate it, skin tenting should always be evaluated!**

c.1.Investigation of the diarrhoeaic calf
c.1.1. Identifying the disease
c.1.1.1. Clinical signs

Acidosis

Historically, metabolic acidosis during calf diarrhoea has been linked primarily with an increase in L-lactic acid and loss of bicarbonate. However, this kind of metabolic acidosis can be compensated through breathing, increased exhalation of carbon dioxide, and through increased metabolism by L-lactase.

However, during the past decade, evidence has accumulated that D-lactic acidosis is a more common occurrence in calves with neonatal diarrhoea. The most probable source of D-lactic acid is bacterial fermentation of undigested substrate that reaches the large intestine due to damage to the mucosal epithelium of the small intestine [29]. This kind of metabolic acidosis is more difficult to compensate due to the absence of D-lactase.

In addition, an acidosis-without-dehydration syndrome in calves is thought to occur during ruminal drinking, with D-lactic acid reported as the cause of this syndrome [30].

EVALUATION OF ACIDOSIS

Precise determination of acidosis with an arterial blood gas test measures the pH directly; otherwise we can evaluate it indirectly by determining the partial pressure of carbon dioxide (see page 48). However, due to the cost of both techniques, neither is usually available to practitioners.

Acidosis is described as a basic deficit of 10-19.9 mEq/L and is considered severe when it reaches 20-30 mEq/L.

! DO NOT CONFUSE...

Acidosis evaluation with dehydration, as we can find severely acidotic calves without diarrhoea and, conversely, calves with extreme dehydration that are not acidotic.

In practice, acidosis is evaluated through three signs:

Ability to stand up

Absence or decrease of suckling reflex

Depression

Geishauser and Thünker [31] describe the relationship between basic deficit and the two behavioural signs of suckling reflex and ability to stand up.

Basic deficit (mEq/L)					
Suckling reflex	Strong	4.2	5.2	Strong	Ability to stand up
	Weak	11.4	7.8	Weak	
	Absent	21.5	19.1	Absent	

Progression of clinical signs from ataxia to coma

Severely acidotic, comatose calf

! TIP

In animals with reduced suckling reflex or depression intravenous treatment is recommended!

c.1. Investigation of the diarrhoeaic calf
c.1.1. Identifying the disease
c.1.1.1. Clinical signs

Septicaemia

Septicaemia is not only a frequent complication of neonatal diarrhoea, but it is also its main differential diagnosis.

Early signs

Early signs indicative of septicaemia are: recumbency, depression, anorexia, absent or decreased suckling reflex, fever or hypothermia, congestion, petechiae or ecchymosis of the sclera.

CAUTION... **We should always check for signs of septicaemia in diarrhoeic calves in order to establish the prognosis and treatment.**

Recumbency and depression in a calf

Scleral congestion

Scleral ecchymosis in a septicaemic calf

TIP **Calves with less than 8% dehydration but with signs of depression will benefit from IV fluids.**

c.1.Investigation of the diarrhoeaic calf
c.1.1. Identifying the disease
c.1.1.1. Clinical signs

Later signs

Signs of localising infection such as hypopyon (infection in the eye), meningitis, pneumonia, diarrhoea, septic arthritis or umbilicus infection.

Hypopyon on the left eye of a septicaemic calf

Umbilicus infection in a calf

Tarsal rear leg septic arthritis in a calf

Paraplegia due to osteomyelitis of a lumbar vertebra secondary to septicaemia in a calf

Meningitis is a frequent and remarkable evolution of the septicaemic picture with altered mental status from depression to mania and coma, tonic-clonic seizures, ocular strabismus, hyperesthesia, and opisthotonus.

Ocular strabismus in a calf with meningitis

Hyperesthaesia and opisthotonus in a calf with meningitis

Finally, after overcoming a severe disease, calves sometimes suffer a loss of hair named anagen defluxion

Anagen defluxion in a calf

c.1. Investigation of the diarrhoeaic calf

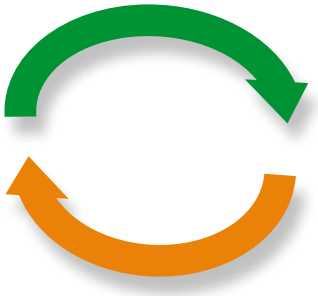
c.1.1. Identifying the disease

c.1.1.1. Clinical signs

Differential diagnosis of the aetiology of septicaemic calves:

DIARRHOEA

20-30% of diarrhoea episodes may develop septicaemia (mainly due to *E. coli)*



SEPTICAEMIA

Septicaemia can cause diarrhoea in the end stage.

Epidemiologic factors and pathology will help you in the differential diagnosis.

Source of septicaemia	Associated with failure of passive transfer of immunity?	Bacteria implicated	Age (days)	Clinical signs
1) Oral/intestinal Umbilical	Yes*	Mainly *E. coli*	2 - 6	Non-specific: depression, anorexia, fever or hypothermia, variable degree of tachycardia and tachypnea, scleral injection and petechiae with rapid progression to death.
2) Secondary to diarrhoea	Partially	Mainly *E. coli*	5 - 20	Usually a less aggressive type with localized infection: arthritis, growth plate, omphalitis, hypopyon, pneumonia, meningitis, etc.
3) Oral, nasal pharyngeal	No	*Salmonella spp*. and *M. haemolytica*	> 4	Similar to *Salmonellosis* and *Mannheimia* pneumonia
4) Secondary to localized infection (navel, joint, growth plate, etc.)**	Partially	Mainly *E. coli*	> 15	Usually a less aggressive type with localised infection: arthritis, growth plate, omphalitis, hypopyon, pneumonia, meningitis, etc.

** Even after sufficient intake of good-quality colostrum, calves may develop septicaemia if bacteria reach the intestine before the colostrum does, which is very frequently the case when pathogen pressure is very high. These cases of septicaemia can develop in an epidemic form.*

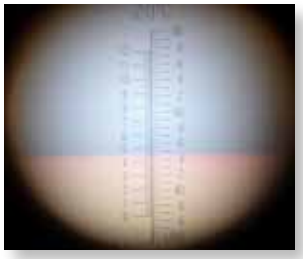
*** Localised infection may be due to previous septicaemia.*

c.1.Investigation of the diarrhoeaic calf
c.1.1. Identifying the disease
c.1.1.2. Additional supporting test : Clinical pathology and biochemistry

Dam immune transfer to the calf

- Determination of IgG level or estimation of IgG level through serum total solids (STS) (see page 99)

Blood collection



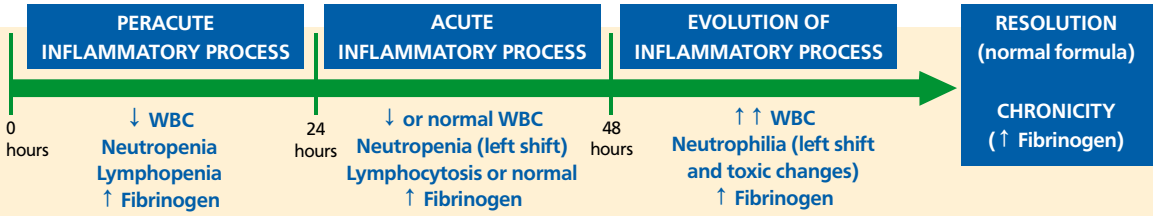
STS < 5.5 g/dl by refractometry of calf serum

Dehydration:

- Packed cell volume (PCV) > 40%
  + elevated STS by refractometry

Septicaemia

- **Haemogram:**
  - Elevated fibrinogen (FB) (>500 mg/dl) or STS:FB ratio lower than 10:1 indicate an active inflammatory process
  - White blood cells:
    - *Total number of white blood cells (WBC)*
      - Increase in inflammatory processes (> 9,000 WBC/µL)
      - Significant increase in severe bacteraemia (12,000-30,000 WBC/µL)
      - Decrease in acute bacteraemia (0-24 h) and viraemia.
    - *Leukogram:*



- **Cerebrospinal fluid (CSF):**

  In suppurative meningitis, CSF shows: high specific density [1.010 (1.007-1.017)]; increased proteins [2.5 g/l (0.5-7.1)]; increased leukocytes [2x109/l (0.012-12.6)], mainly neutrophils; and reduced glucose.

- **Blood culture:** definitive diagnosis of septicaemia requires blood culture

CAUTION... **We should always check for signs of septicaemia in diarrhoeaic calves in order to establish the prognosis and treatment.**

TIP **Always consider both relative and absolute values in the differential white cell count!!**

c.1.Investigation of the diarrhoeaic calf
c.1.1. Identifying the disease
c.1.1.2. Additional supporting test : Clinical pathology and biochemistry

Electrolyte disturbances

Vary among individual cases.

- Hyponatraemia is usually present due to faecal loss.
- Hyperkalaemia is often observed in very acidotic calves, even though total potassium concentration actually decreases. Such hyperkalaemia probably occurs when potassium translocates from intracellular to extracellular compartments.

Calves should always have free access to fresh and clean water

DO NOT CONFUSE...

Blood electrolyte levels can vary considerably due to oral and intravenous fluid administration → It is very important that calves have access to water to compensate for these changes.

DO NOT CONFUSE...

Hypernatraemia due to treatment with oral electrolytes can occur when calves do not have free access to fresh water!

Metabolic acidosis:

- Direct determination of venous gases: arterial blood gas sampling. Portable blood gas analyser, such as the I-Stat unit (Heska Corporation, Fribourg, Swizerland) determine blood gas and acid-base status. However, it is expensive. Other, less expensive methods are the use of a portable pH meter (Cardy Twinn pH meter, Spectrum Technologies, Plainfield, EE.UU.) or the Harleco-System to determine total carbon dioxide concentration.
- To determine levels of D-lactate with stereospecificity, high-performance liquid chromatography or enzymatic assays are necessary. A portable lactate analyzer exists, but it measures only L-lactate.
  - **DIRECT DETERMINATION:**
    - The pH is low (<7.35)
    - Bicarbonate levels are decreased (<20 mEq/L).
    - Excess of base (negative, <0 mEq/L)
  - **INDIRECT DETERMINATION:**
    - CO_2 concentration in plasma (<26 mEq/L)
    - Strong ion gap

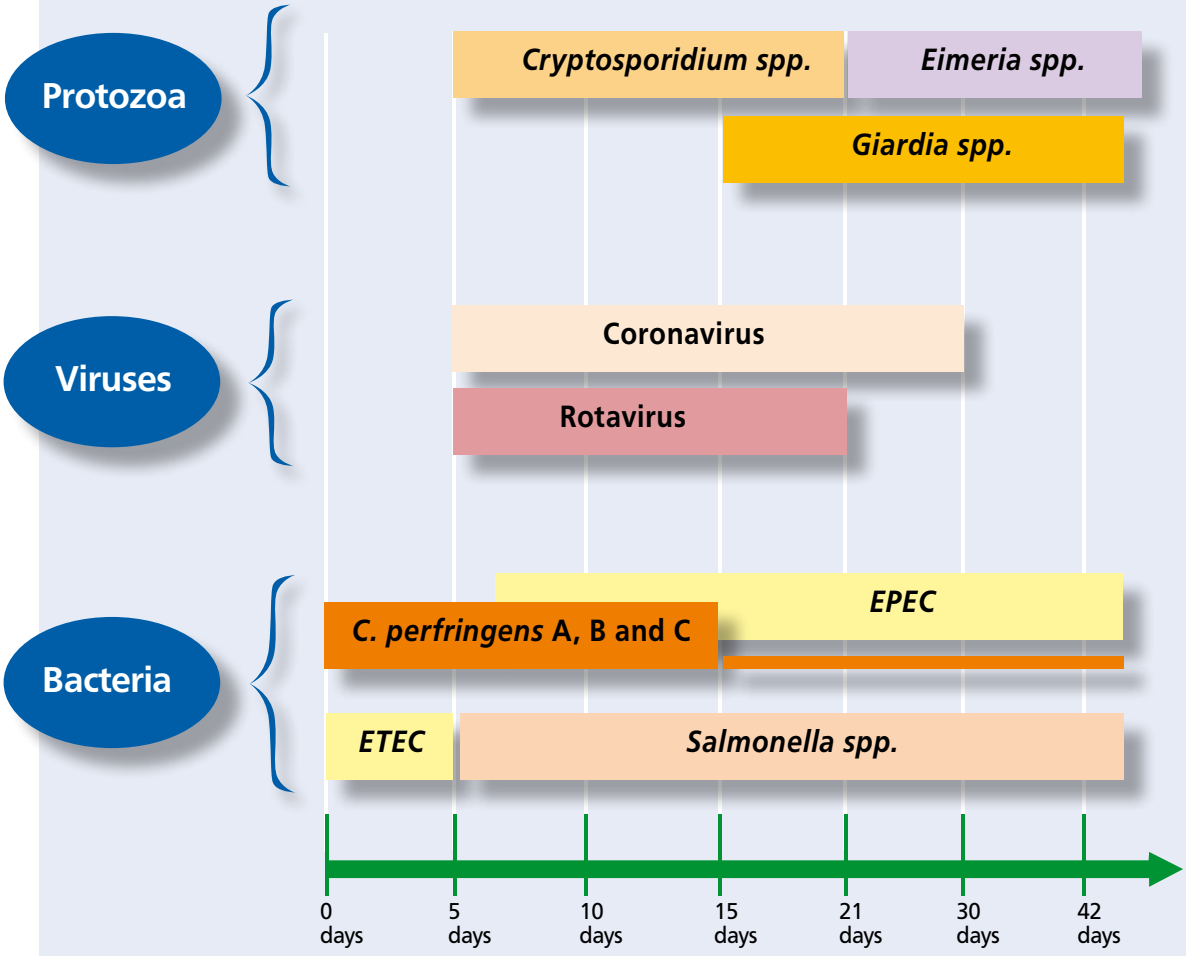
c.1.Investigation of the diarrhoeaic calf
c.1.2. Identifying the aetiology
c.1.2.1. Clinical signs by pathogen

Clinical signs caused by different aetiologies of neonatal diarrhoea in calves.

Aetiology			Age	Systemic signs	Most frequent appearence of faeces
Non-infectious	Nutritional	Osmotic diarrhoea	Variable	In chronic cases: cachexia, growth retardation, recumbency and inability to get up without help	Voluminous white clay faeces
		Ruminal drinking	Variable	Acidosis, cachexia and dehydration	White clay faeces
		Diarrhoea due to hypoproteinaemia	Variable	Cachexia, growth retardation, recumbency and inability to get up without help	Scarce diarrhoeaic faeces
Infectious	Bacteria	*E. coli (ETEC)*	1 - 15 days	Fever-normothermia-hypothermia. Accumulation of liquid in abdomen → sucussion splashing	Profuse, liquid and yellowish
		Salmonella spp.	4 - 28 days and older	Fever and depression. Can be complicated with septicaemia	Dysentery with mucus, fibrin casts and foul odour
		C. perfringens types A, B and C	1 - 15 days and older	Colic pain. Endotoxaemia. Sudden death	A → Sometimes with melaena B and C → Haemorrhagic enteritis
	Viruses	Rotavirus	5 - 21 days	Fever in some cases.	Thick, whitish and with mucus → progression to liquid
		Coronavirus	5 - 30 days	Death in 72 hours due to hypovolaemia if not treated	Yellow- to blood-stained mucus-containing diarrhoea
	Protozoa	*Cryptosporidium spp.*	4 - 20 days	Mild fever in some cases	Yellowish and liquid
		Eimeria spp.	> 3 weeks	Without fever. Sometimes with tenesmus	Initially gray → progression to dysentery
		Giardia spp.	> 15 days	Usually no systemic effects	Temporary, mild diarrhoea; aqueous or gelatinous yellow with mucus or blood

c.1.Investigation of the diarrhoeaic calf
c.1.2. Identifying the aetiology
c.1.2.1. Clinical signs by pathogen

Figure. Age of susceptibility for the main enteropathogens involved in infectious neonatal diarrhoea.



c.1.Investigation of the diarrhoeaic calf
c.1.2. Identifying the aetiology

c.1.2.1. Clinical signs by pathogen

UNDIFFERENTIATED NEONATAL DIARRHOEA:

Based on clinical findings alone, it is often not possible to differentiate between the most common pathogens involved (ETEC, rotavirus, coronavirus, Crypstosporidium spp....; or frequently mixed infections). Undifferentiated neonatal diarrhea involves different enteropathogens, frequently as mixed infections. Clinical findings are not pathognomonic. Laboratory testing is needed to confirm the enteropathogen involved.

c.1.2.1.1. Enterotoxigenic *Escherichia coli* (ETEC)

DIARRHOEA:
Abdominal distension with splashing sounds at ballottement → voluminous, watery diarrhoea (coating the tail, perineum and hind legs).

SYSTEMIC:
Initial fever (40° C) → normothermia → hypothermia.

Dehydration → weakness and coma hypovolaemic shock within hours after onset.

Severe metabolic acidosis.

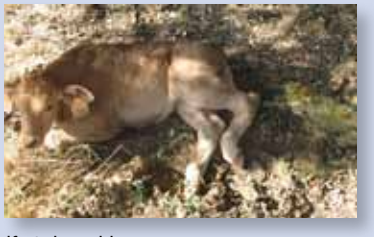
Watery faeces characteristic of secretory diarrhoea in a 3-day-old calf

Dehydrated and weak calf with watery diarrhoea

c.1.2.1.2. Rotavirus and Coronavirus

DIARRHOEA:
Initially thick, white-yellowish and with mucus → progression to liquid in 72 h.
Sometimes fresh blood is present.

SYSTEMIC:
Depression, dehydration and acidosis (D-lactate).



White-yellowish pasty-syrup faeces in a calf 10 days old

c.1.2.1.3. *Cryptosporidium parvum*

DIARRHOEA:
Yellowish and moderately liquid but very persistent; peak occurs at 3-5 days after infection and lasts 4-17 days.

SYSTEMIC:
Sometimes mild fever.

Yellowish pasty faeces in a calf 15 days old

Yellowish liquid faeces in a calf 20 days old

c.1.Investigation of the diarrhoeaic calf
c.1.2. Identifying the aetiology
c.1.2.1. Clinical signs by pathogen

c.1.2.1.4. *C. perfringens B and C*

DIARRHOEA:
Peracute cases have abdominal distention without diarrhoea coming out of the animal. Acute cases involve disentery with abdominal pain

SYSTEMIC:
Progressive dehydration, depression, abdominal distention and shock. May cause colic or convulsions and death in less than 12 hours.

C. perfringens A

DIARRHOEA:
Low in volume.

SYSTEMIC:
Signs of tympany and colic precede diarrhoea. Progressive dehydration, depression, abdominal distention anaemia and shock.

Dysentery caused by *C. perfringens*

c.1.2.1.5. *Salmonella spp.*

DIARRHOEA:
Dysentery with mucus, fibrinous, caseous material in faeces, pseudomembranes and foul odour

SYSTEMIC:
Fever, anorexia, depression and severe systemic signs due to septicaemia → septic physitis, peritonitis, arthritis, meningitis and pneumonia may be present.
Distal gangrenous necrosis of extremities (pinnae, digits, tail).
Dehydration and metabolic acidosis.

Dysentery

Distal gangrenous necrosis of calf extremities can be found

Faecal dysentery staining of hind quarters

Outbreak of salmonellosis

c.1.2.1.6. *Eimeria spp.*

DIARRHOEA:
Gray and pasty but very persistent. In some cases fresh blood is present.
Faecal staining of hind quarters.

SYSTEMIC:
Tenesmus and rectal prolapse may be present.
No fever, but acidosis is severe.
Nervous coccidiosis causes tremor, hyperaesthesia, opisthotonus, convulsions, nystagmus and recumbency.

Rectal prolapse

Gray and pasty persistent diarrhoea

Diarrhoea with fresh blood

c.1.Investigation of the diarrhoeaic calf
c.1.2. Identifying the aetiology
c.1.2.1. Clinical signs by pathogen

c.1.2.1.7. *Giardia spp.*

DIARRHOEA:
Temporary, mild aqueous or yellow gelatinous diarrhoea with mucus or blood. Diarrhoea can persist or recur over time.

SYSTEMIC:
Not usually.

Mild aqueous gelatinous diarrhoea

c.1.2.1.8. Osmotic diarrhoea

DIARRHOEA:
Voluminous white clay faeces.

SYSTEMIC:
Not usually.

Clay faeces

c.1.2.1.9. Ruminal drinking

DIARRHOEA:
White clay faeces.

SYSTEMIC:
Acidosis, cachexia.

Dehydration.

Clay faeces in a calf

c.1.2.1.10. Diarrhoea due to hypoproteinaemia

DIARRHOEA:
Scarce diarrhoeic faeces.

SYSTEMIC:
Cachexia, growth retardation, recumbency and inability to get up without help. Although in recumbency, the calf maintains its appetite.

Cachectic calf with diarrhoea with strong sucking reflex

Weak cachectic calf unable to stand up unaided

c.1.Investigation of the diarrhoeaic calf
c.1.2. Identifying the aetiology
c.1.2.2. Supporting test by pathogen

Blood collection

Clinical pathology and biochemistry by aetiology

	Agent		Clinical pathology	Biochemistry
Non-infectious	**Nutritional**	Ruminal drinking	Leucogram frequently shows leucocytosis with high levels of fibrinogen	Metabolic acidosis
		Hypoproteinaemia	Hypoproteinaemia	Biochemistry may be normal
Infectious	**Bacteria**	*E. coli (ETEC)*	Dehydration higher than 8% → elevation of haematocrit and proteins while WBC counts remain usually normal	Mild hyponatraemia and hypochloraemia are inconsistently present Hypoglycaemia
		Salmonella spp.	Classical picture consists of a degenerative left shift with neutropaenia and band neutrophilia. Although blood is present in faeces, anaemia is masked by dehydration. Despite dehydration, total proteins are usually normal or low because of their loss into the gut and malabsorption	Metabolic acidosis Hyponatraemia and hypochloraemia Potassium may range from high to low depending on severity and duration of diarrhoea
		C. perfringens types A, B and C	Haemoconcentration (increased hematocrit and proteins) Leucogram is normal	Biochemistry may be normal
	Viruses	Rotavirus	Dehydration higher than 8% → elevation of hematocrit and proteins WBC usually normal	Not specific enough to help in diagnosis, as results vary with severity and duration of disease. Severely affected calves develop metabolic acidosis and low plasma bicarbonate. Other electrolytes and glucose tend to be low
		Coronavirus		
	Protozoa	*C. parvum*	Mild dehydration	Biochemistry may be normal
		Eimeria spp.	Dehydration and anaemia, which can be masked by dehydration	Hyponatraemia

c.1.Investigation of the diarrhoeaic calf
c.1.2. Identifying the aetiology
c.1.2.3. Lesions at necropsy *(pathomorphology)*

Lesions found at necropsy help with differential diagnosis of aetiology of neonatal diarrhoea. Identifying the aetiology is important as it allows implementation of appropriate prophylactic and therapeutic measures.

From lesions to causes:

- **Non-infectious:**
  - Ruminal drinking
  - Diarrhoea due to hypoproteinaemia by cachexia

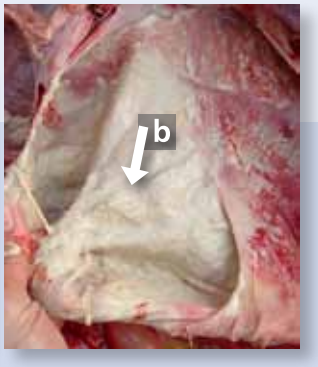
- **Infectious causes:**
  - Undifferentiated neonatal diarrhoea (ETEC, rotavirus, coronavirus, *Cryptosporidium spp...)*
  - *Salmonella spp.*
  - *C. perfringens*
  - *Eimeria spp.*
  - Septicaemic *E. coli*
  - EPEC

c.1.Investigation of the diarrhoeaic calf
c.1.2. Identifying the aetiology

c.1.2.3. Lesions at necropsy *(pathomorphology)*

c.1.2.3.1. Ruminal drinking

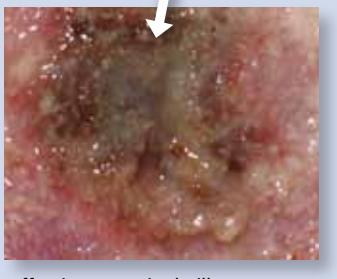
When ruminal drinking is present, no gross lesion will be found, apart from the abnormal presence of milk inside the rumen. However, milk fermentation in the rumen produces volatile fatty acids and lactic acid, leading to a decrease in pH that may cause rumenitis and/or enteritis.



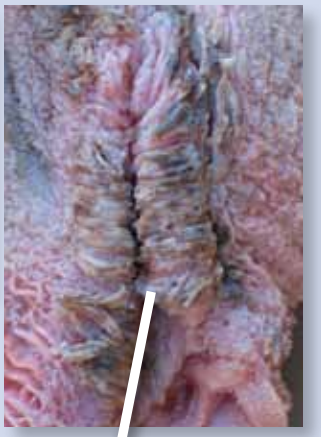
Abnormal presence of milk in the rumen, with a liquid (a) or clay (b) appearance.

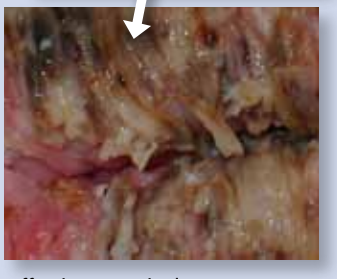
Absence of gross lesions in abdominal and thoracic cavities

Rumenitis due to decreased rumen pH can be found



Affecting a ruminal pillar





Affecting a ruminal groove

Jejunal mucosa with irritative enteritis due to acid content

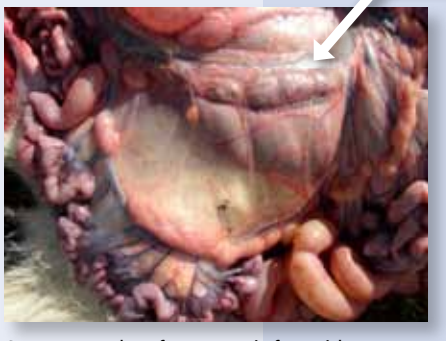
c.1.2.3. Lesions at necropsy *(pathomorphology)*

c.1.2.3.2. Diarrhoea due to hypoproteinaemia

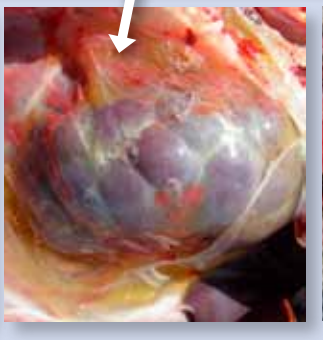
Hypoproteinaemia due to cachexia will cause mobilisation of fat deposits, and serous atrophy will be the most characteristic gross lesions found.

Cachectic calf

Serous atrophy (gelatinous masses substitute fat deposits)

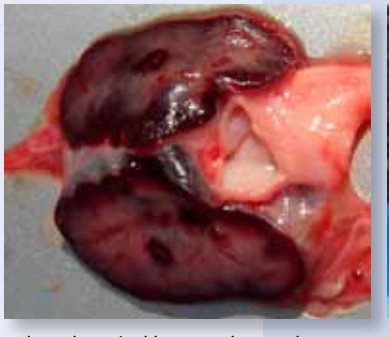


Serous atrophy of mesenteric fat, with dilatation of mesenteric lymphatic vessels with perilymphatic cuffs



Serous atrophy in perirenal fat

Serous atrophy in epicardial fat



Adrenal cortical haemorrhages due to chronic stress

Subcutaneous oedema caused by hypoproteinaemia

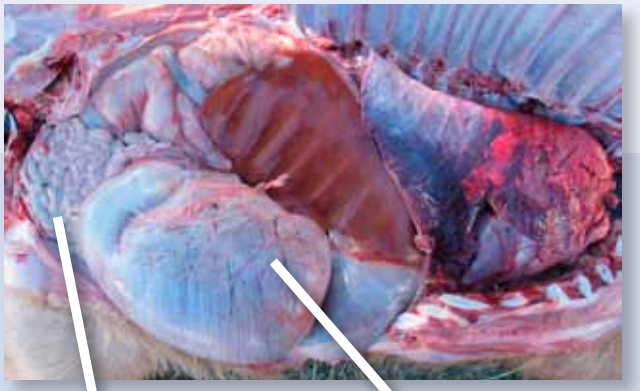
In premature weaning or lack of adaptation to concentrated feed, the rumen is full of undigested feed

c.1.Investigation of the diarrhoeaic calf
c.1.2. Identifying the aetiology

c.1.2.3. Lesions at necropsy *(pathomorphology)*

c.1.2.3.3. Undifferentiated neonatal diarrhoea

At necropsy different causes of neonatal diarrhoea cannot be differentiated without laboratory assistance, as little or no morphologic change may be evident.



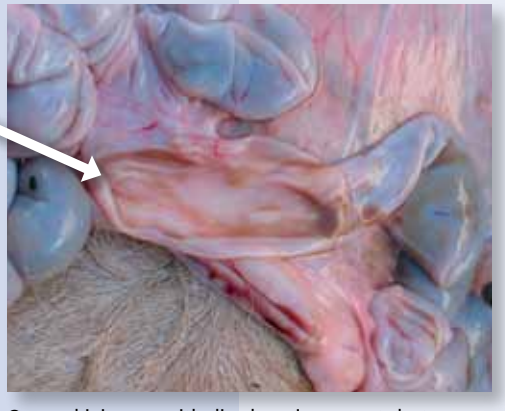
Abdominal cavity that lacks visible gross lesions (there are no signs of septicaemia or toxaemia), and a large quantity of liquid contents are retained in the digestive tract

Distended abomasum

Mesenterium and jejunum without visible gross lesions and full of abundant liquid content

DO NOT CONFUSE...

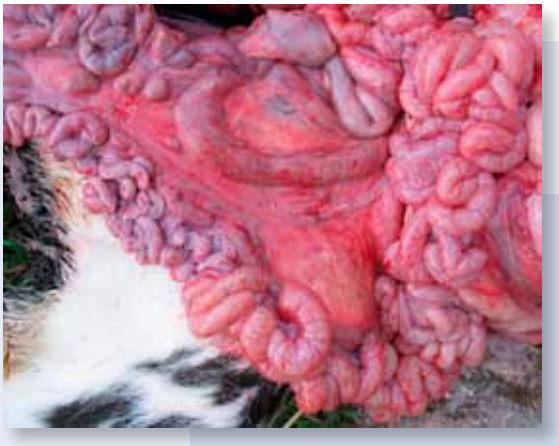
This kind of diarrhoea does not produce lymph node enlargement, but young calves show larger lymph nodes than older ones



Opened jejunum with diarrhoeaic contents but with no visible gross lesions

c.1.Investigation of the diarrhoeaic calf
c.1.2. Identifying the aetiology

c.1.2.3. Lesions at necropsy *(pathomorphology)*



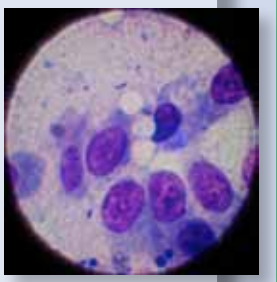
A congestive appearance over the entire abdominal cavity can be observed in some cases of undifferentiated neonatal diarrhoea

Reddish color of the distal part of the mucosal fold is a postmortem artefact, but true colonic lesions are typical of coronavirus infection. Confirmation must be done by histopathology

In chronic cases, allopecia in the perianal area is a very frequent finding

Laboratory techniques to confirm the enteropathogen involved in undifferentiated neonatal diarrhoea

Pathogen	Laboratory methods to identify enteropathogen involved
ETEC	Histological examination of small intestine sections shows the absence of a severe villous atrophy and presence of bacteria on the surface of villi. Specific immunofluorescent techniques. Culture and characterisation of pathogenic factors.
Coronavirus and rotavirus	Histological examination shows villous atrophy. Demonstration of virus in faeces. Demonstration of viral antigen in infected epithelium.
C. parvum	Histological examination shows villous atrophy characterised by blunting and fusion of villi and hypertrophy of crypts of Lieberkühn. *Cryptosporidium* can be recognised in the brush border of villi in the small intestine epithelium and in ileal mucosal smears, as well as in faecal smears or faecal flotation.



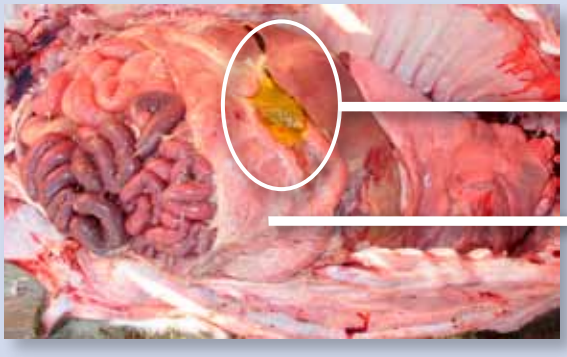
C. parvum in an ileal mucosa smear

c.1.Investigation of the diarrhoeaic calf
c.1.2. Identifying the aetiology

c.1.2.3. Lesions at necropsy *(pathomorphology)*

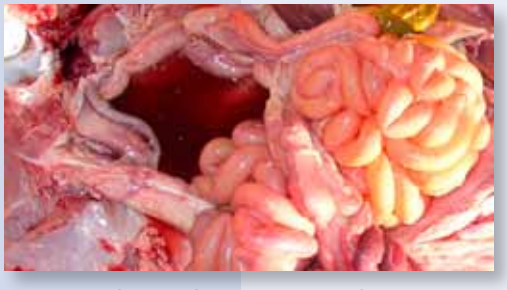
c.1.2.3.4. *Salmonella spp.*

Salmonellosis causes diffuse haemorrhagic enteritis with fibrinous enterocolitis, in which intestinal contents are very characteristic. In addition, gallbladder inflammation is a pathognomonic lesion of the disease.

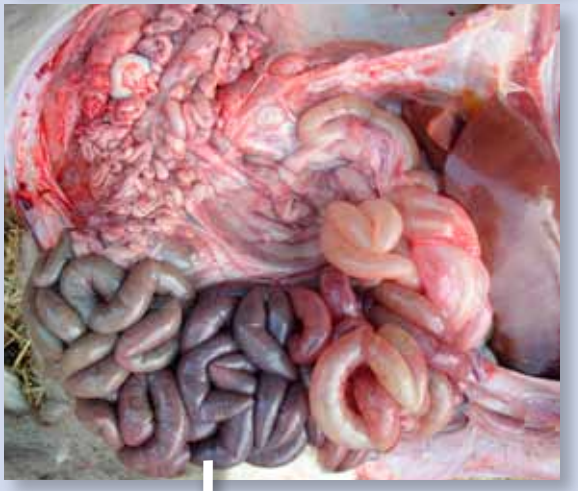


Inflammation of gallbladder with histological fibrinous cholecystisis is considered pathognomonic of acute enteritis due to *Salmonella spp.*

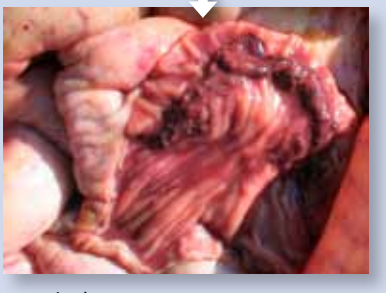
Fibrinous peritonitis (only present in a proportion of cases with septicaemia)



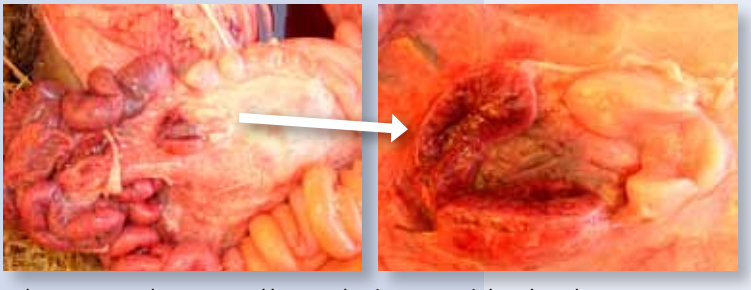
Serohaemorrhagic contents in the abdominal cavity



Diffuse catarrhal haemorrhagic enteritis with fibrinous enterocolitis



Intestinal contents are watery, malodorous and may contain mucus or whole blood with typical casts



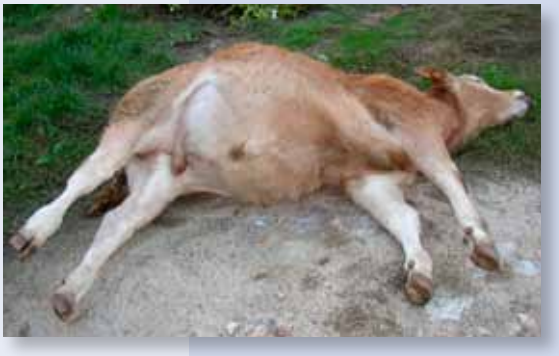
Enlargement, oedematous and haemorrhagic mesenteric lymph nodes

c.1.Investigation of the diarrhoeaic calf
c.1.2. Identifying the aetiology

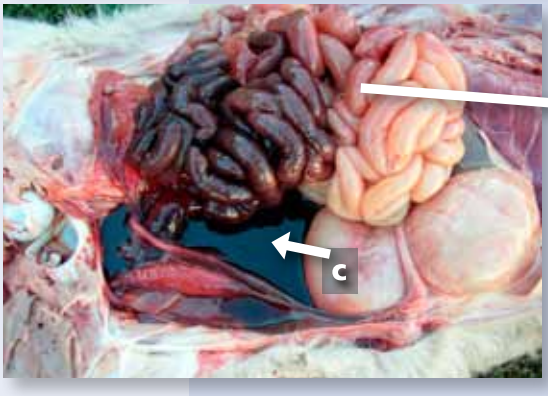
c.1.2.3. Lesions at necropsy *(pathomorphology)*

c.1.2.3.5. *C. perfringens* types B and C

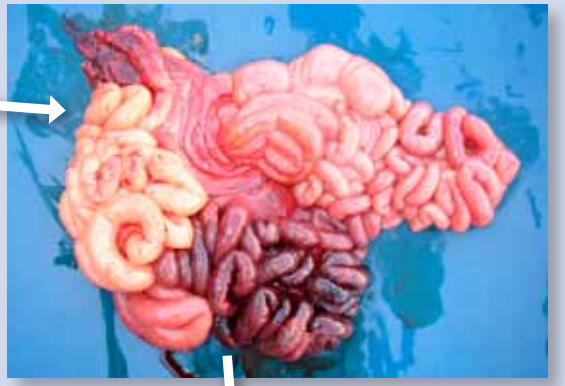
In infections of *C. perfringens* types B and C, necrotic-haemorrhagic enteritis involving the larger or smaller part of the jejunum is characteristic. However, confirmation of the diagnosis requires detection of the pathogenic toxins produced by *Clostridium spp.*



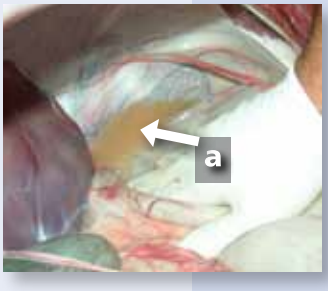
Tympanitic calf typically found in clostridial diseases

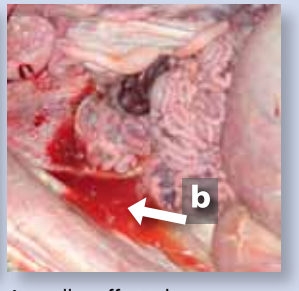


The colour of ascitic liquid can vary from amber (a) or reddish (b), to the most common black-reddish (c)

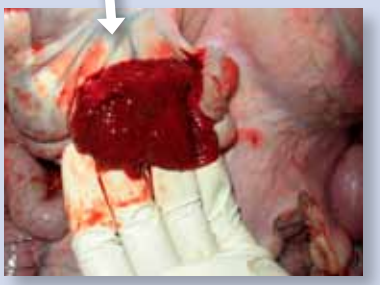


Necrotic-haemorrhagic enteritis involving the jejunum





A smaller affected area can be indicative of *C. perfringens B*



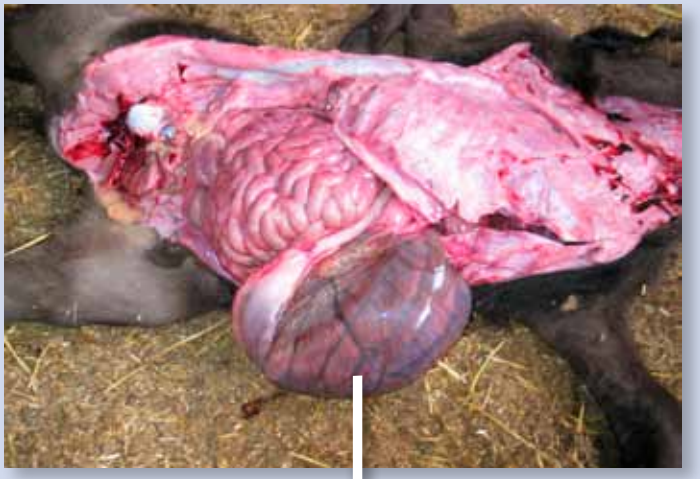
Necrotic-haemorrhagic jejunal mucosa with bloody contents

c.1.Investigation of the diarrhoeaic calf
c.1.2. Identifying the aetiology

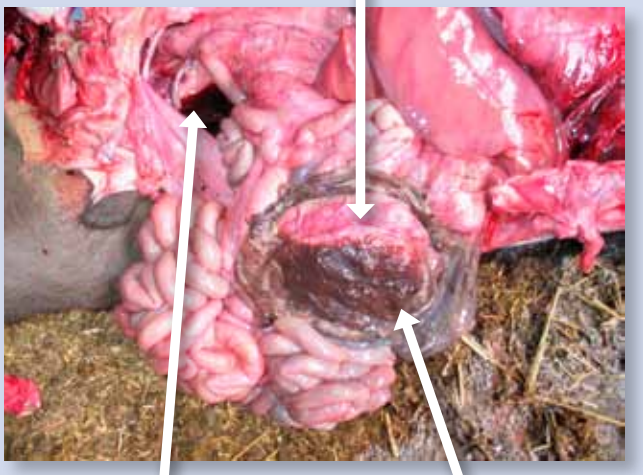
c.1.2.3. Lesions at necropsy *(pathomorphology)*

c.1.2.3.6. *C. perfringens* type A

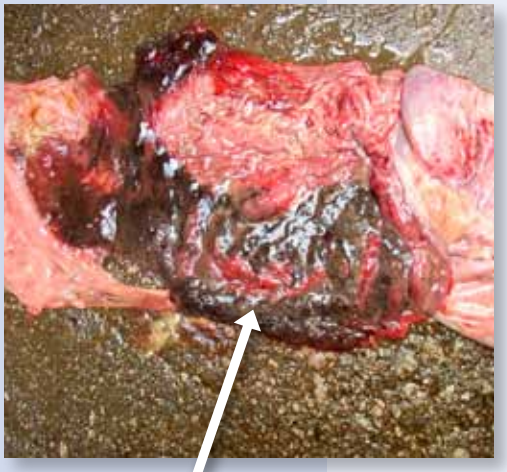
In *C. perfringens* type A infection, the main gross lesion present is an enlarged and oedematous abomasum. There are also reports of a fatal hemolytic disease and an acute hemorrhagic enteritis associated with the presence of large numbers of *C. perfringens* A in the intestine. Confirmation of diagnosis requires the detection of the pathogenic toxins produced by the clostridium.



Abdominal cavity without visible gross lesion except for a very distended and oedematous abomasum



Haemorrhagic ascitic liquid



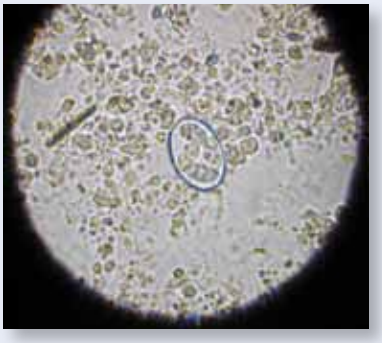
Haemorrhagic and oedematous abomasal mucosa

c.1.2.3. Lesions at necropsy *(pathomorphology)*

c.1.2.3.7. *Eimeria spp*. (coccidiosis)

Coccidiosis causes a typical fibrinohemorrhagic typhlocolitis. In addition, rectal prolapse may be present. Diagnostic confirmation can be achieved through oocyst detection in mucosal scrapings or by faecal flotation.

Rectal prolapse is a characteristic sign of coccidiosis

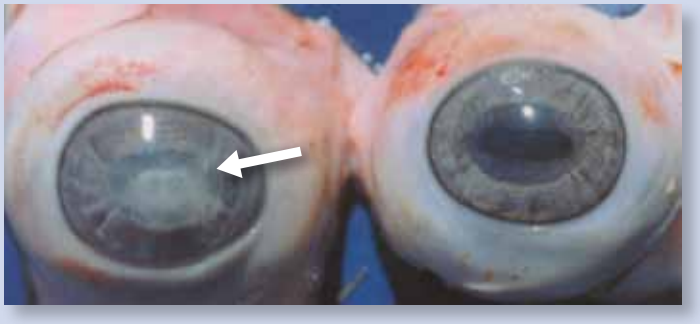


Confirmed at necropsy by finding a large number of developmental stages in mucosal scrapings or identifying the oocysts by faecal flotation

Typical fibrinohaemorrhagic typhlocolitis, which may extend to the rectum.
The mucosa is oedematous with exaggerated longitudinal folds
Contents are abnormally fluid and vary from brown to black with mucus

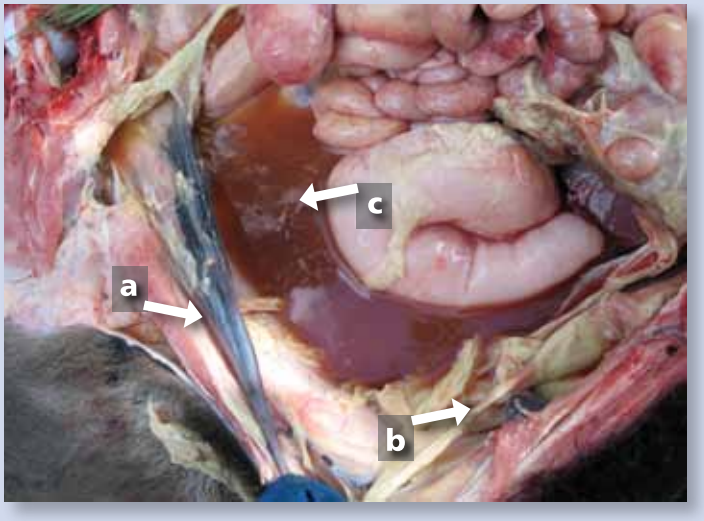
c.1.2.3.8. Septicaemia

Main cause of neonatal septicaemia is digestive septicaemic *E. coli*, which typically causes arthritis and hypopyon.



Hypopyon

Septic arthritis



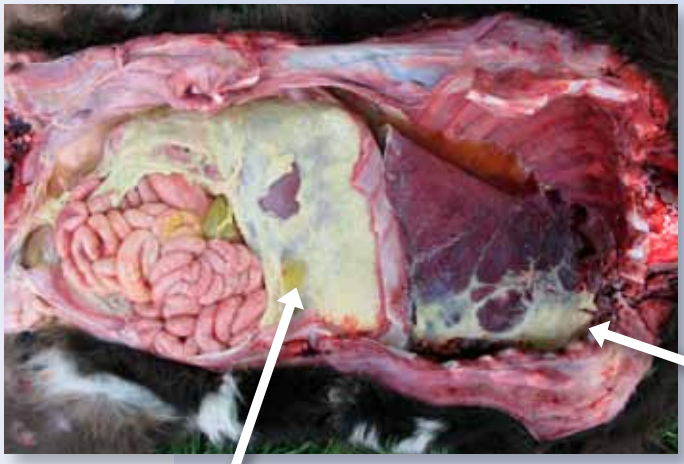
Umbilical infection is the second most common cause of septicaemia: omphaloarteritis (a) and omphalophlebitis (b) with peritonitis (c)

c.1.Investigation of the diarrhoeaic calf
c.1.2. Identifying the aetiology

c.1.2.3. Lesions at necropsy *(pathomorphology)*

c.1.2.3.8. Septicaemia

Fibrin is always a sign indicative of septicaemia.

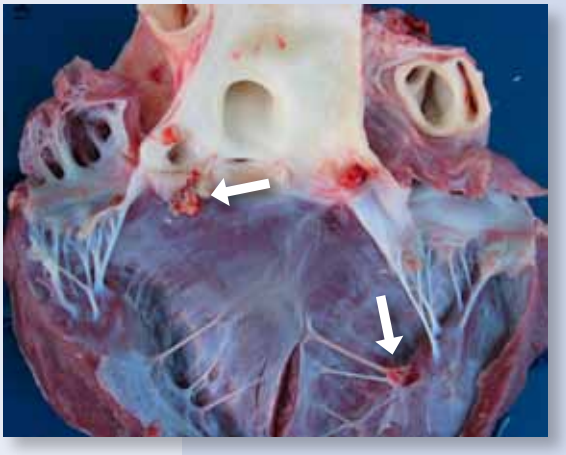


Peritonitis

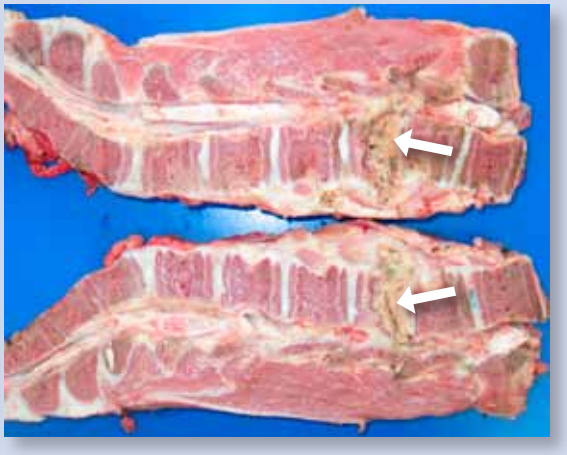
Pleuritis

Pericarditis

Localised lesions of septicaemia such as endocarditis or osteomyelitis can give rise to secondary septicaemia in the future.



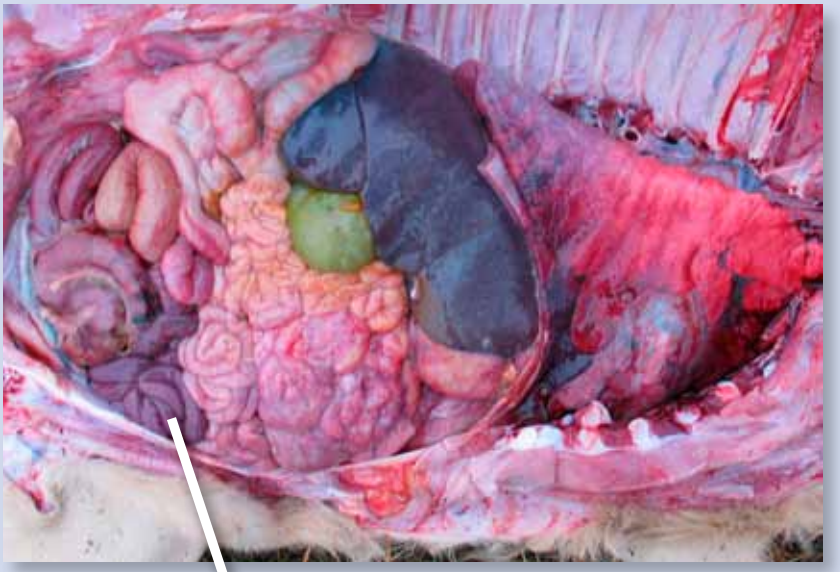
Endocarditis



Osteomyelitis (spondylitis)

c.1.2.3.9. Enteropathogenic *E. coli* (EPEC)

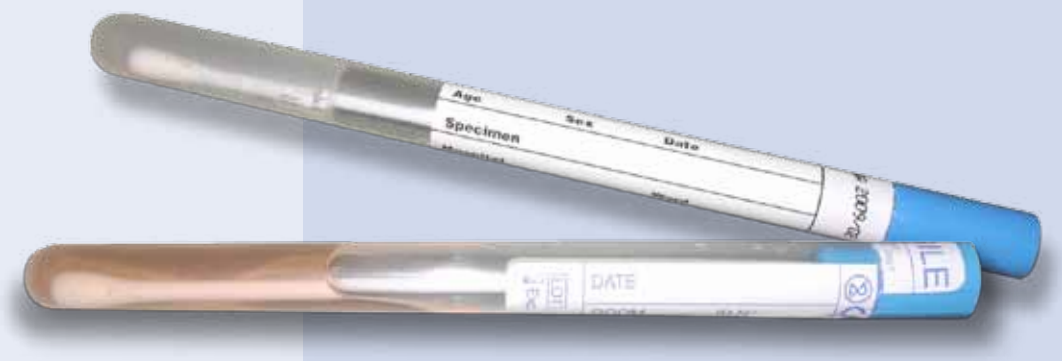
Erosive fibrino-haemorrhagic enterocolitis with peritonitis and lung congestion is characteristic of EPEC. However, pathogenicity factors should be identified to confirm the diagnosis.



Erosive fibrinohaemorrhagic enterocolitis with peritonitis and lung congestion

c.1.Investigation of the diarrhoeaic calf
c.1.3. Looking for the pathogens

c.1.3.1. Which samples should be taken



c.1.3.1. Which samples should be taken

Animals with clinical symptoms:

- **Rectal swab:**
  - Bacteria + + +
  - Virus + + +
  - Parasites +/-

- **Rectal faecal sampling:**
  - Virus + + +
  - Bacteria + + +
  - Parasites + + +

Collect samples from about 10% of clinically symptomatic calves (no fewer than three)

Legend:

+ : poor matrix
+ + : adequate matrix
+ + + : good matrix

c.1.Investigation of the diarrhoeaic calf
c.1.3. Looking for the pathogens

c.1.3.1. Which samples should be taken

Organs:

- **Abomasum:**
  - Bacteria: *Clostridium spp.* + + + (not indicated for other bacteria)
  - Virus +
  - Parasites + + +

- **Small Intestine:**
  - Bacteria + + +
  - Virus + + +
  - Parasites + + +

- **Large Intestine:**
  - Bacteria + +
  - Virus +
  - Parasites + + +

c.1.3.2. Sampling procedures

Rectal swabs:

- **Material**
  Swab with transport media
- **Procedure**
  Introduction in the anal sphincter
- **Analysis**
  Pathogens
  Virus: good matrix
  Bacteria: very good matrix
- **Technique**
  Bacteriology: Cultures
  Virology: Tissue cultures (cytopathic effect)
  Swab positions Electron microscopy
  and PCR

NOTE
Samples should be taken at the early onset of diarrhoea

Rectal faecal sampling:

- **Material**
  sterile gloves
- **Procedure**
  Introduction of the finger in the anal sphincter
- **Analysis**
  Pathogens
  Virus: very good matrix
  Bacteria: good matrix
  (pay attention to possible non-specific contamination)
- **Technique**
  Bacteria: On-site test
  (lateral immunochromatography) *E. coli*
  Culture
  Virology: On-site test
  (lateral immunochromatography) Rotavirus,
  Coronavirus
  ELISA antigene test
  Tissue cultures (cytopathic effects)
  PCR
  Electron microscopy
  Parasitology: On-site test by lateral immunochromatography for *C. parvum*
  Coprology (flotation with saturated solutions)

c.1.3.2. Sampling procedures

Organs:

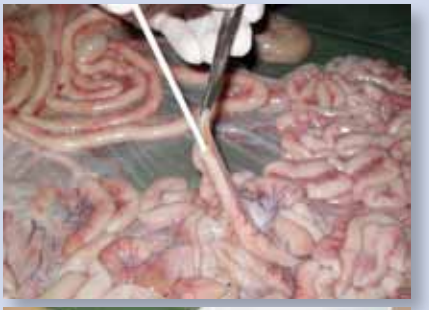
SMALL INTESTINE

- **Location:**
  - Jejunum with sterile swabs (bacteriology-virology)
  - Complete section of the whole wall (histology)
  - Intramural lymphatic structures (Peyer's patches) (Histology-virology)
  - Mesenteric lymph nodes (histology-bacteriology)

- **Analysis:**
  - Bacteriology: Pathogenic *E. coli*, *Clostridium perfringens* types A, B, C. *Clostridium sordelli, Salmonella enterica* serotypes (Dublin, Typhimurium and Newport).
  - Virology: Rotavirus, Coronavirus, Calicivirus, Torovirus, BVDV
  - Parasitology: Protozoa *(Eimeria spp., Cryptosporidium spp, Giardia spp.)*, Nematodes (Ascaridia, Strongyloides, Strongyles)

The anatomic-pathologic picture can help determine the pathophysiology:

No lesions
Catarrhal enteritis
Haemorrhagic enteritis
Pseudo-membranous enteritis
Necrotic-ulcerative enteritis



Small intestine sterile swabs (jejunum)

Mesenteric Lymph nodes

Peyer's patches

c.1.3.2. Sampling procedures

Organs:

LARGE INTESTINE

- **Where to collect:**

  From the colon using sterile swabs (bacteriology-virology)

  Removal of a section of the whole wall (i.e. histology)

- **Analysis:**

  - Bacteriology: *Salmonella enterica serotypes (*Dublin, Typhimurium and Newport), some strains of *E. coli* (EHEC)
  - Virology: Coronavirus
  - Parasitology: Protozoa *(Eimeria spp., Cryptosporidium spp., Giardia spp.)*, Nematodes (Ascaridia, Strongyloides, Strongyles)

The anatomic-pathologic picture can help determine the pathophysiology:

Haemorrhagic enteritis

Pseudo-membranous enteritis

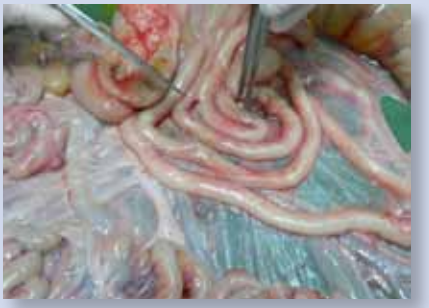
Necrotic-ulcerative enteritis

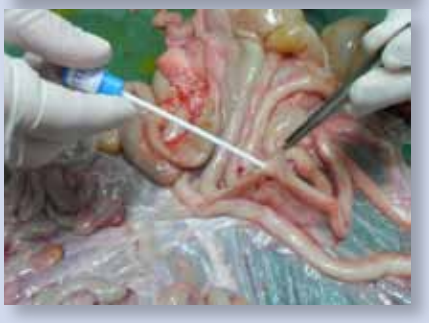
Aetiological orientation based on anatomic-pathological lesions:

Pseudo-membranous enteritis: salmonellosis

Haemorrhagic enteritis: *Clostridium spp.*

Necrotic-ulcerative enteritis: BVDV





c.1.3.2. Sampling procedures

Blood:

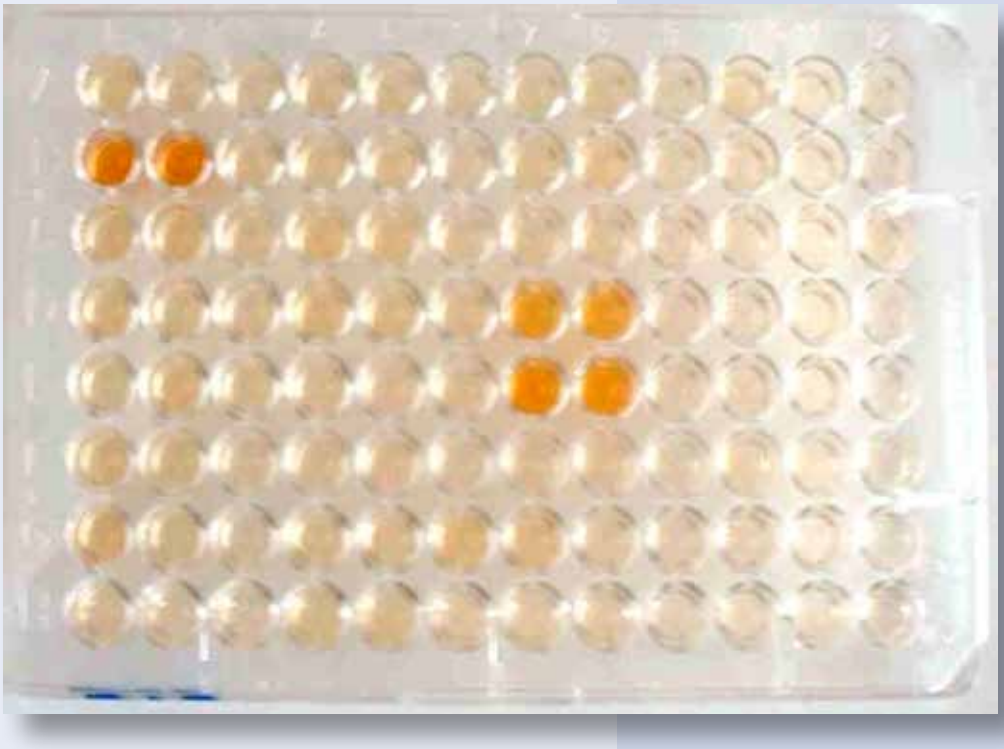
- **Serology:**
  - Techniques:

    ELISA, serum neutralisation test, indirect immunofluorescence Test (IFI).

    In general serology is not routinely used, because of interference with maternal immunity

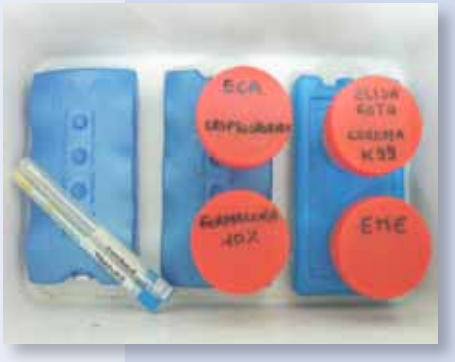
- **Blood cultures:**

  To diagnose septicaemic germ



c.1.3.3. Shipping of the material

Time frame (from sampling to analysis):



- Ideal: < 6 hours
- Time limit: < 48 hours

  Note: for the organs, the time between death and sampling or analyzing has to be taken into account):
  - To be modulated according to pathogens:
    - Rotavirus are more resistant than other viruses
    - Bacteria and parasites are more stable
    - Clostridia: take into account fast replication post mortem

Interpretation of *C. perfringens* concentration in the intestinal contents [32]

CFU / ml of intestinal contents	*Time between death and sampling (in hours)*			
	< 3 h	<3 < t < 6 h	< 6 < t < 15 h	> 15 h
$< 10^5$	Negative	Negative	Negative	Not valuable
$< 10^5 < n < 10^6$	Doubt	Doubt	Doubt	Not valuable
$< 10^6 < n < 10^7$	Positive	Doubt	Doubt	Not valuable
$< 10^7 < n < 10^8$	Positive	Positive	Doubt	Not valuable
$> 10^8$	Positive	Positive	Positive	Not valuable

Sampling techniques:

- Rectal sample: within 12 hours
- Rectal faecal sample: viruses - within 12 hours
- Bacteria: within 12 - 24 hours
- Parasites: within 24 - 48 hours

Analysis techniques:

- Bacteriological examination: within 24 hours
- Virus isolation from tissue cultures: within 12 hours
- Viral antigen identification by PCR: within 24-48 hours
- Parasitological test: within 48-72 hours

c.1.3.3. Shipping of the material

Shipping conditions:

- Refrigeration (0 to 4°C);
  Virus: 6 - 12 hours
  Bacteria: 12 - 24 hours
  Parasites: up to 7 days
- Freezing (-20°C)
  Freezing bacteria, viruses and parasites drastically reduces the possibility of isolating them.

Transport media:

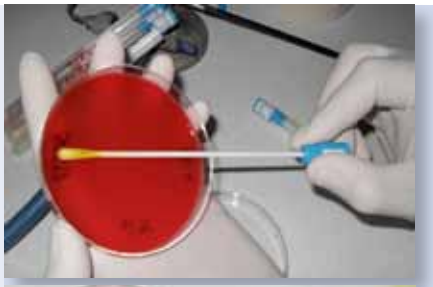
- Not essential if the time frame and temperature of shipping are strictly adhered to.
  Bacteria: transport culture swabs

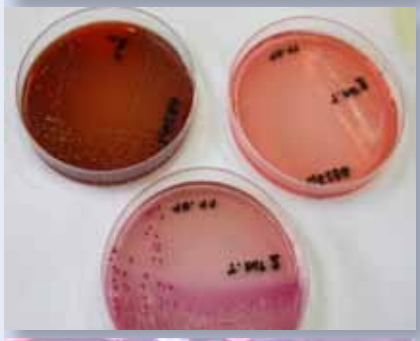
Virus isolation in tissue culture: transport media with antibiotics

Time to obtain results

- Bacteria: - Identification in culture: 3-5 days
- Viruses: - Cultures from cell tissues: 5-30 days
  - Immunofluorescence (IF): a few hours
  - Electron microscopy: 1-2 days
  - ELISA antigens: a few hours
  - PCR: 8-24 hours
- Parasites: - Qualitative tests by flotation technique: 1-2 days
  - Quantification of eggs or oocysts (McMaster technique): 1-2 days

c.1.3.4. Laboratory tests





Bacteria

- **Techniques**
  Routine Cultures Pathogenic *E. coli* strains, *Clostridium perfringens* type A, B, C. *Clostridium sordelli, Salmonella enterica* serotypes Dublin, Typhimurium, and Newport.

Important: characterization of the *E. coli* strains, toxin typing of clostridia and sero-typing of salmonellae

- **Sampling**
  Rectal samples, rectal faecal samples, Organs:+++
  Animals treated with antibiotics can give false negative results
- **Shipping of the material**
  Within 24 hours at controlled temperatures (0- 4 °C)
  Use of transport media is not necessary if the shipping time frame and temperature are respected
- **Interpretation of results**
  Results must be interpreted according to the pathogenic power of the bacteria and of the observed lesions.
  More frequent pathogens: Pathogenic *E. coli* strains , *Clostridium perfringens* type A and C, *Salmonella enterica* serotypes Dublin and Typhimurium.
  Less frequent pathogens: *Clostridium perfringens* type B, *Clostridium sordelli, Salmonella enterica* Newport.
- **Antibiogram**
  Antibiograms are important for monitoring possible antimicrobial resistance (*E. coli* in particular)

Techniques: the Kirby-Bauer method is used routinely

This technique cannot be used for aerobic pathogens, in which case specific alternative methods must be used (MIC).

Interpretation
Take care that the sampled material is representative: sampled animals should be representative of the observed clinical signs and preferably untreated.

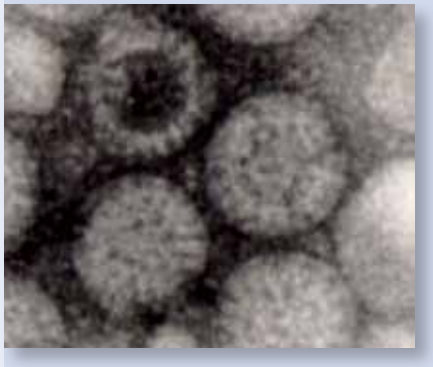
c.1.Investigation of the diarrhoeaic calf
c.1.3. Looking for the pathogens

c.1.3.4. Laboratory tests

Viruses

Major viral pathogens:

Rotavirus

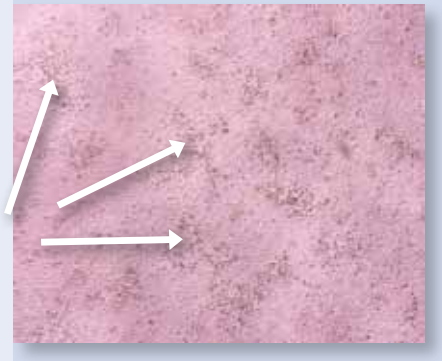


- **Peculiarity:**
  Virus strictly cell-bound
  Double capsid virus without envelop
  Virus particularly resistant in the environment

- **Techniques:**
  Serology: ELISA test, virus neutralisation test (VN), Immunofluorescence Inhibition (IFI)

  Virology: isolation on tissue cultures

  Antigen detection:
  - Good sensitivity
  - Immunofluorescence (IF)
  - Immuno-enzymatic tests, antigen ELISA)
  - Electronic microscopy (on fresh samples)
  - PCR (performed in specialist laboratories)



- **Sampling:**
  Rectal samples, rectal faecal samples, ORGANS: +++

- **Shipping:**
  Refrigeration temperature (0 - 4 °C)
  It is possible to freeze the samples if the time between shipping and the test is >12 hours

NOTE

It is preferable to sample live animals as autolysis and bacterial invasion occur within 5 minutes after death

Proper storage and shipping of samples is important for success of subsequent isolation on tissue cultures and detection with electronic microscopy

c.1.3.4. Laboratory tests

Coronavirus

- **Peculiarity:**
  Coronavirus can be involved in "Winter Dysentery" in adult cattle and in respiratory outbreaks in calves
  Enveloped RNA virus
  Coronavirus is less resistant than Rotavirus in the environment

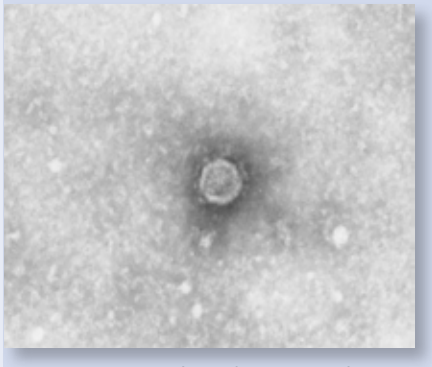
- **Techniques:**
  Serology: ELISA test, virus neutralisation test (VN), IFI.

  Virology: isolation on tissue cultures

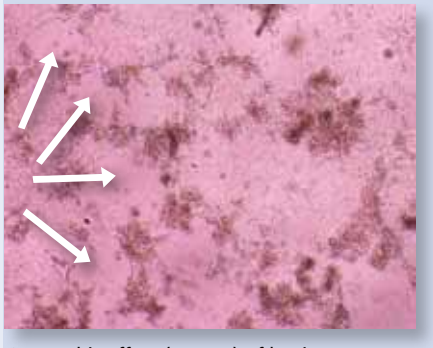
  Antigen detection:
  - Good sensitivity
  - Immunofluorescence (IF)
  - Immuno-enzymatic tests, antigen ELISA)
  - Electronic microscopy (on fresh samples)
  - PCR (performed in specialist laboratories)

- **Sampling:**
  Rectal samples, rectal faecal samples, ORGANS: +++

- **Shipping:**
  Refrigeration temperature (0 - 4 °C)
  It is possible to freeze the samples if the time between shipping and the test is >12 hours.



Bovine coronavirus (2009/45 TN strain)
Electronic microscopy X 75.000



Cytopathic effect (arrows) of bovine coronavirus (2009/45 TN strain), in tissue culture (Optical microscopy, 40X).

c.1.3.4. Laboratory tests

Other viruses (Calicivirus, Torovirus, BVDV)

- **Peculiarity:**
  Virusses less stable than Rotavirus and Coronavirus

- **Techniques:**
  Serology: ELISA test, VN

  Virology: isolation on tissue cultures.
  Low sensitivity for Torovirus (BToV)

  Antigen detection:
  - Good sensitivity
  - Immunofluorescence (IF)
  - Immuno-enzymatic tests, antigen ELISA)
  - Electronic microscopy (on fresh samples)
  - PCR (performed in specialist laboratories)

Histology:
Collect a section of tissue and put into a container containing a suitable fixative (10% buffered formalin solution). Send the material to a specialized laboratory.

- **Sampling:**
  Rectal samples, rectal faecal samples, ORGANS: +++

- **Shipping:**
  Refrigeration temperature (0 - 4 °C)
  It is possible to freeze the samples if the time between shipping and the test is >12 hours.

c.1.3.4. Laboratory tests

Parasites

Major parasites:

Cryptosporidia

- **Peculiarity:**
  Oocysts are very resistant in the external environment. Possibility of autoinfection through the presence of sporulated, thin-walled oocysts in the intestinal lumen.

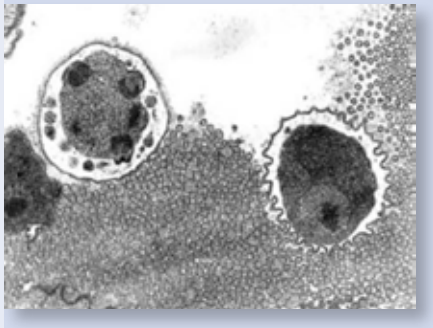
- **Techniques:**
  Coprological test (flotation in saturated solutions) and staining with Ziehl-Neelsen.

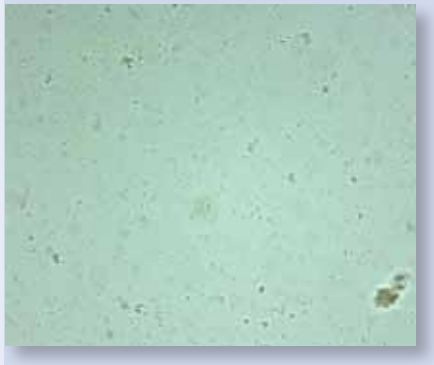
  Antigen ELISA test
  Immunoflourescence

  PCR
  Electron microscopy
  Immunochromatography
  Histology

- **Sampling:**
  Rectal samples, rectal faecal samples, ORGANS: +++

- **Shipping:**
  Refrigeration temperature (0 - 4 °C)

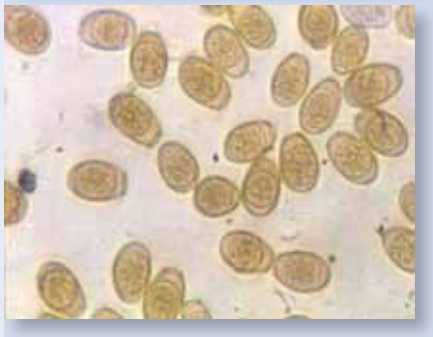




c.1.3.4. Laboratory tests

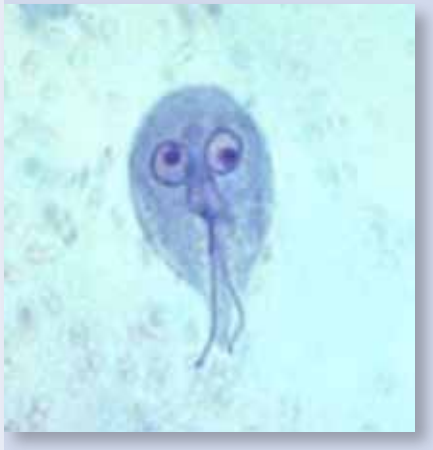
Coccidia: (Eimeria spp.)

- **Peculiarity:**
  Oocysts are very resistant in the external environment.
- **Techniques:**
  Coprological test (flotation in saturated solutions)
- **Sampling:**
  Rectal faecal samples, ORGANS: +++
- **Shipping:**
  Refrigeration temperature (0 - 4 °C)



Giardia spp.

- **Peculiarity:**
  Unicellular flagellated parasites able to infect different hosts, including humans. Oocysts are very resistant in the environment.
- **Techniques:**
  Microscopy on fresh samples from intestinal contents (detection of trophozoites)
  On faeces: Antigen ELISA test, PCR
- **Sampling:**
  Rectal faecal samples, ORGANS: +++
- **Shipping:**
  Refrigeration temperature (0 - 4 °C)



c.1.3.4. Laboratory tests

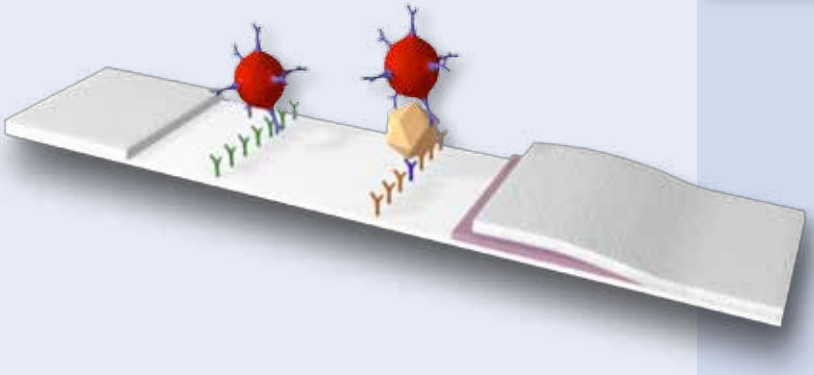
Other parasites
(Strongyloides, Ascaridia, Strongyles)

- **Peculiarity:**
  Eggs pollute the environment and are not susceptible to common disinfectants
- **Techniques:**
  Coprologic test (flotation in saturated solutions)
- **Sampling:**
  Rectal samples, rectal faecal samples, ORGANS: +++
- **Shipping:**
  Refrigeration temperature (0 - 4 °C)

Histology:
Collect a section of tissue and put into a container containing a suitable fixative (10% buffered formalin solution). Send the material to a specialized laboratory.

c.1.3.5. On-site tests

Chromatographic lateral flow immunoassay available as on-site tests for diagnosis of neonatal diarrhoea.



Kits designed to detect

- Rotavirus
- Coronavirus
- *E. coli* F5 (K99)
- *C. parvum*

Easy to use, no laboratory equipment required, incubation at room temperature, results within 10 minutes

c.2.1. Steps along the investigation

1. Identify the problem

At least 3 months of exact, retrospective data should be reviewed to evaluate the significance and evolution of the outbreak.

From the data try to establish the at-risk age group and other epidemiologic characteristics.

Finally, examine calves with typical characteristics of the problem and sample the faeces of a minimum of 3 calves.

2. Locate the source of the problem by evaluating risk factors

As transmission is mainly faecal-oral, the source of the problem is usually manure present in: maternity pen bedding, contaminated colostrum, calf pen bedding, contamination of feed, on dam udder etc.

3. Characterise the factors that create susceptibility

This requires an evaluation of herd management practices, especially those related to the dam, calf and colostrum, which can give rise to a failure of passive immune transfer; herd management practices related to biosecurity should also be evaluated.

PRACTICAL TIP

Age of diarrhoea onset varies for different enteropathogens, which will help us establish a first differential diagnosis

PRACTICAL TIP

Knowledge of the enteropathogens involved will help us better define sources and sites of exposure

c.2. Investigation of an outbreak

c.2.2. Template for data collection on the farm

Farm Information:
Name of the farmer:

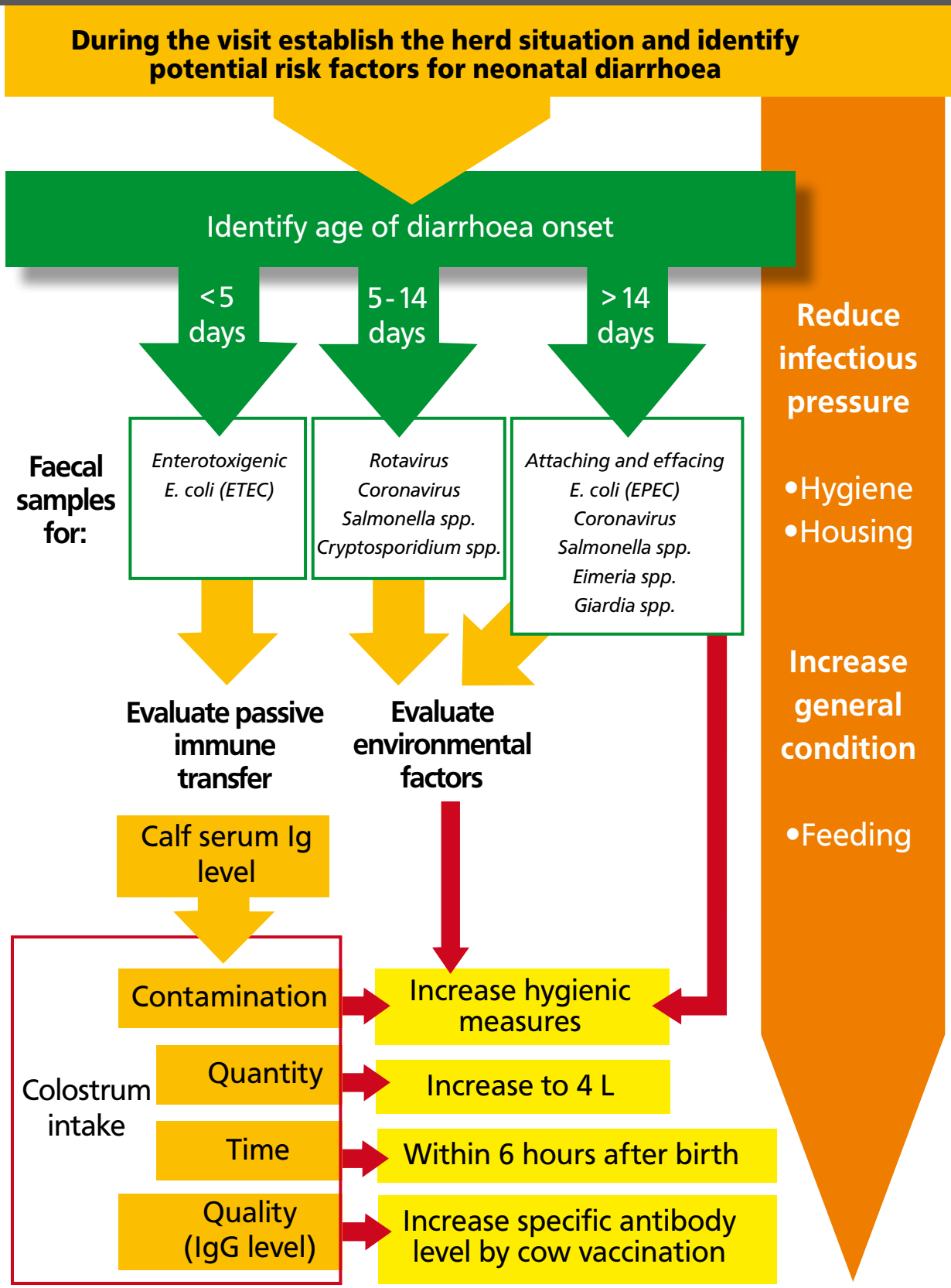
Visit Information:
Farm history done by: _____ Date of first farm visit:

General management of the farm	Number of animals	Cows	Cows in lactation					
			Cows in dry period					
		Calves	0-1 year old					
			1-2 years old					
	Personnel	Who takes care of the calves?						
	Housing	Calving and calves	Calving: Individual calving box / calving dry area / communal calving area					
			How long are calves with their dams?					
			Type of calf housing: __ Individual boxes (material:_____) __ Group boxes (how many:_____) __ Group pen (how many: _____)					
			How many calf pens?					
			Do calves have direct contact with each other? NO / YES					
			Do calves have visual contact with others? NO / YES					
		Hygienic measures	Removal of calving area bedding between calvings?					
			Farm-specific clothing? NO / YES					
			Specific order of handling? NO / YES (____ from young to old, _____ other (specify: _____)					
					Calving area		Calf pen	
			Cleaning.	When?				
				How often?				
			Disinfection	When?				
				How often?				
				Products used				
	Feeding	Colostrum	Suckled or collected and administered?					
			Interval between calving and milking?					
			Interval between birth and first colostrum intake?					
			Nipple bottle or feeding tube?					
			Volume of colostrum at first meal?					
			Timing and volume of subsequent meals?					
			Quality of colostrum evaluated? NO / YES					
			Colostrum bank? NO / YES					
			Pasteurisation of colostrum? NO / YES Tª/time?					
		Milk Replacer	Commercial name?					
			Composition?					
			Preparation?					
		Feeding practices	Age group	Cow milk	Milk replacer	Roughage	Pellets	Water
			0 - 5 days					
			5 - 14 days					
			14 - 30 days					
			Older					
		Cleaning and disinfection of buckets, nipple bottles, etc.?						
		Does each calf have his own bucket? NO / YES						

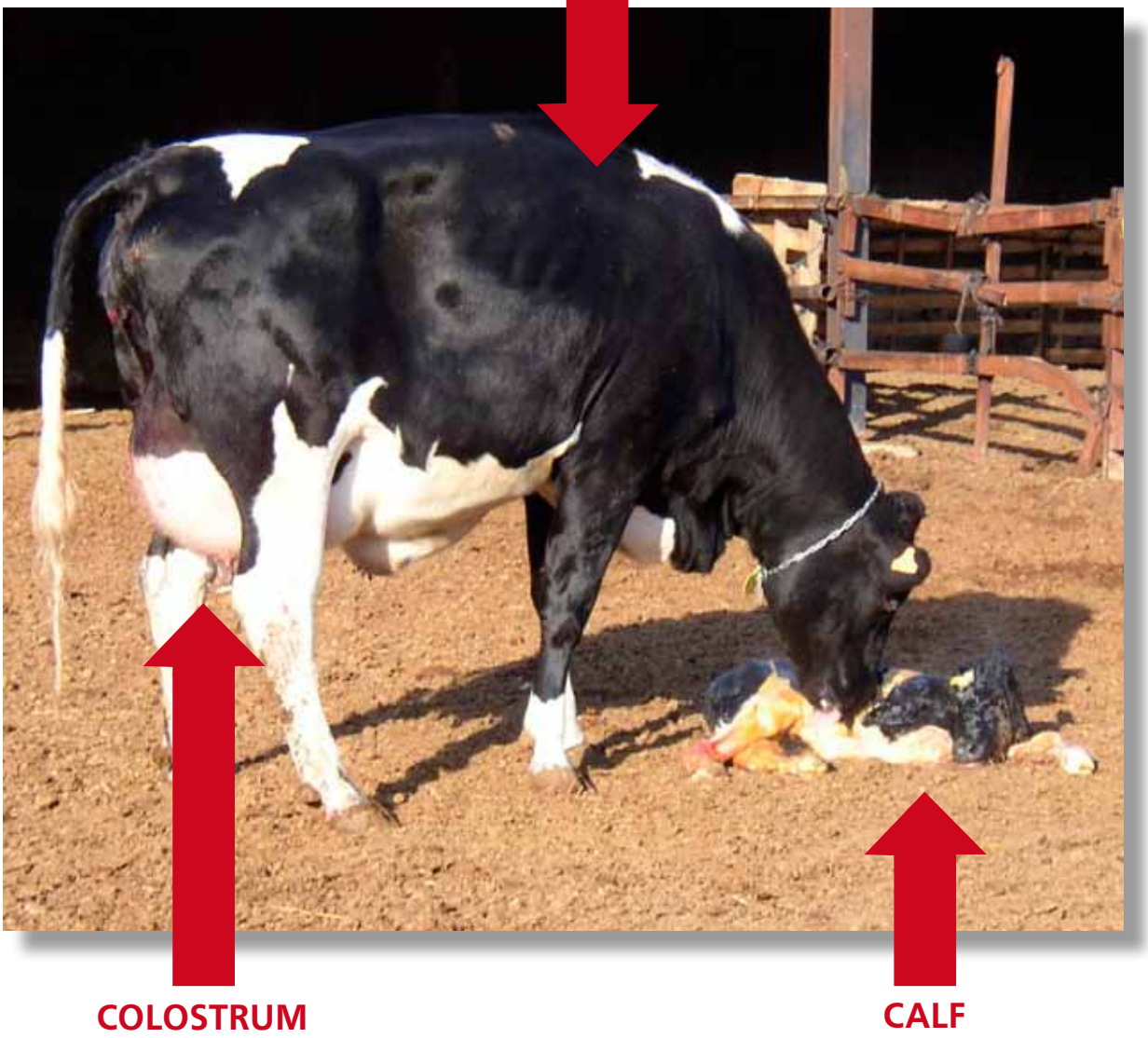
Template for data collection on the farm (continued)

Disease situation	Herd status	BVD				
		Salmonella				
		Others				
	Neonatal diarrhoea problem	Since when?				
		Age group affected	Diarrhoea level	48h - 5 days	5 -21 days	Older
			No diarrhoea			
			Mild (still eating)			
			Moderate (off food)			
			Severe (depressed)			
			Death			
		Severeness and characteristics of diarrhoea:				
		Systemic signs	NO / YES			
			If YES, which ones: depression, acidosis, dehydration, septicaemia, others?			
	Previous laboratory results	Calf immune transfer	Serum refractometry: _____(no. calves STS<5.5mg/dL)/_____Total			
			Immunoglobulin (Ig) G: _____ (no. calves IgG<10mg/L)/_____ Total			
		Coprology				
		Faecal cultures				
	Post Mortem Examination					
	Measures	Specific measures applied to improve neonatal health				
		Medication (electrolytes, antibiotics, other)				
		Dry cow vaccination				
		Treatment results				

c.2. Investigation of an outbreak



d.1. Identifying good practices to ensure dam-to-calf immune transfer



d.1. Identifying good practices to ensure dam-to-calf immune transfer

d.1.1. The dam

d.1.1.1. Before colostrum collection

Factors we can influence

- **DRY PERIOD:**
  - Feeding (vitamins and minerals)
  - A length of 45-60 days is recommended
    - <40 dry days leads to reduced volume (2.2 kg less) [33]
    - <25 dry days leads to reduced Ig concentration [34]
- **HEAT STRESS:**
  - Reduces dry matter intake, and this is associated with poor colostrum composition (lower IgG, IgA, total protein, casein, fat and lactose)
  - Reduces calf weight
- **PRE-PARTUM MILKING or milk loss** (Ig are stored at this time, and will be lost through milking)
- **PRE-PARTUM VACCINATION** against neonatal diarrhoea pathogens increases specific colostral antibodies against *E. coli*, rotavirus and coronavirus (see page 108)

Individual maternity pen

Dry cow yard

Factors we cannot influence

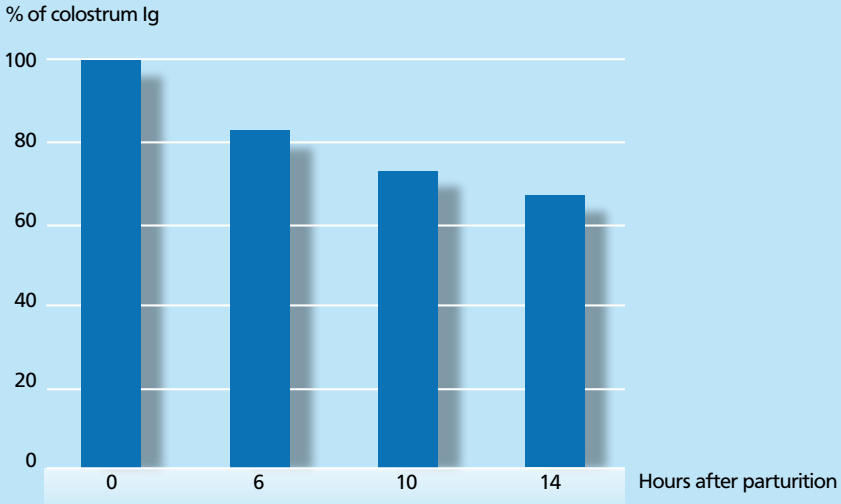
- **DAM AGE** (Ig content increases with age, probably due to the greater immunological experience)
- **BREED** (A dilution effect has been observed: breeds with higher colostrum production have lower Ig concentrations)
- **SEASON AT PARTURITION** (Studies give contradictory results about the effect of season on colostrum quality; the conflicting findings may be related to heat stress)

d.1. Identifying good practices to ensure dam-to-calf immune transfer

d.1.1. The dam

d.1.1.2. At colostrum collection

- The maximum Ig concentration is achieved immediately after calving and decreases over time if collection is delayed [35]
- Colostrum should be collected fewer than 6 hours after calving



Resorption of immunoglobulins when colostrum collection is delayed after parturition reduces colostrum Ig content [35]

In addition, use of the following types of colostrum should be avoided:

- Mastitic
- Watery or bloody
- From cows that leaked before calving
- From cows that were milked pre-partum

Portable milking machine to collect colostrum

PRACTICAL TIP

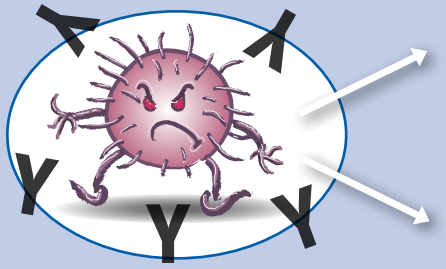
A portable milking machine is useful to avoid waiting until milking time for colostrum collection; in contrast, waiting to milk the cow in the milking parlour will often delay colostrum collection by 6-12 hours.

d.1. Identifying good practices to ensure dam-to-calf immune transfer

d.1.2. The colostrum

d.1.2.1. Colostrum contamination and pasteurisation

Negative effects of colostrum contamination



Potential pathogenic effect: transmission of pathogens from dam to the calf

These pathogen-Ig complexes interfere with immunoglobulin absorption

SOURCES OF COLOSTRUM CONTAMINATION

- Improper practices
  - Inadequate udder preparation
  - Inadequate sanitation or malfunction of milking machine
  - Inadequate cleanliness of milk collection bucket
- Inadequate cooling or storage: avoid storing at room temperature
- Mastitis and other diseases
- Inadequate cleaning of calf nipples or feeding tubes

PRACTICAL TIP

A portable milking machine is useful for reducing colostrum contamination

Hygiene of portable milking equipment is of the utmost importance

d.1.2.2. Pasteurisation of colostrum

- **PASTEURISATION IS RECOMMENDED WHEN**
  Colostrum cannot be handled in an aseptic way, it must be pooled, or it must be taken from cows infected by paratuberculosis, salmonellosis or mycoplasmosis

- **TYPE OF PASTEURISATION**
  An approach using lower temperature for longer time (60 minutes in a 60° C water bath) is a safe way to preserve the Ig and limit bacterial growth

- **EFFECTS OF PASTEURISATION**
  - Elimination or significant reduction of important pathogens: *E. coli, Salmonella spp., Mycoplasma bovis,* or *Mycobacterium avium* subsp. *paratuberculosis*
  - In addition, the shelf life of the pasteurised, refrigerated colostrum is extended to 10 days.
  - Improves lgG absorption

Device for colostrum pasteurisation at the herd level

d.1. Identifying good practices to ensure dam-to-calf immune transfer

d.1.2. The colostrum

d.1.2.3. Storage of colostrum

- Good-quality colostrum should be stored refrigerated or frozen in 2-litre plastic bottles
- Storage is recommended to solve different herd problems: low-quality colostrum of a dam, death of a dam at parturition, etc.
- The shelf life of stored colostrum depends on the type of storage:
  - **REFRIGERATED** **7 DAYS** (unpasteurised colostrum)
    10 DAYS (pasteurised colostrum)
  - **FROZEN** **6 MONTHS**
- A good idea would be to classify the colostrum based on its quality, in order to use the best one for the first meal and the other ones for subsequent meals
- Raw colostrum must not be pooled, as it compromises good-quality colostrum, and it poses a high biosecurity risk

Storage of colostrum in 2-L bottles

Frozen colostrum

Frozen colostrum

Thawing a 5-L colostrum bottle in a water bath

HEATING AND THAWING STORED COLOSTRUM

- Colostrum must be warmed to 35 - 40° C before calf feeding
- The warming must be done in a bain-marie (water bath)
- Overheating (>60° C) destroys the antibodies

CAUTION... **To avoid overheating, do not use the microwave oven or hot water from the milking parlour cleaner system (80-90°C) to thaw or heat colostrum!!!**

d.1.3. The calf

d.1.3.1. Minimising causes of stress

Visual contact is important to reduce stress

At birth, the specific immune system of a calf is only partly functional. Moreover, the immunity can be impaired by:

- Stress factors such as cold, absence of visual contact with other calves or the dam, hypoglycaemia, etc.
- Vaccination before the age of five days [36] or vaccination that does not follow a correct schedule can be counter-productive.

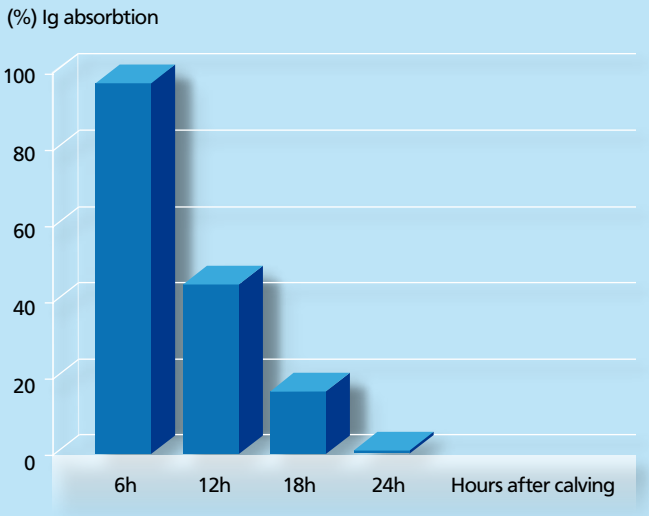
d.1. Identifying good practices to ensure dam-to-calf immune transfer

d.1.3. The calf

d.1.3.2. Time to first feeding

- **Optimal time is the first 4 h after calving.**
- **Efficiency of absorption decreases from 6 h post calving onwards.**
- **After 24 h, Ig will work only in the intestinal lumen.**

Absorption of Ig from colostrum into the blood stream is time-dependent



Ig absorption decreases very rapidly after calving, reaching nil at 24 hours [37]

CAUTION... **Delaying the calf's first feeding is one of the most frequent causes of insufficient levels of colostral antibodies in the calf.**

d.1. Identifying good practices to ensure dam-to-calf immune transfer

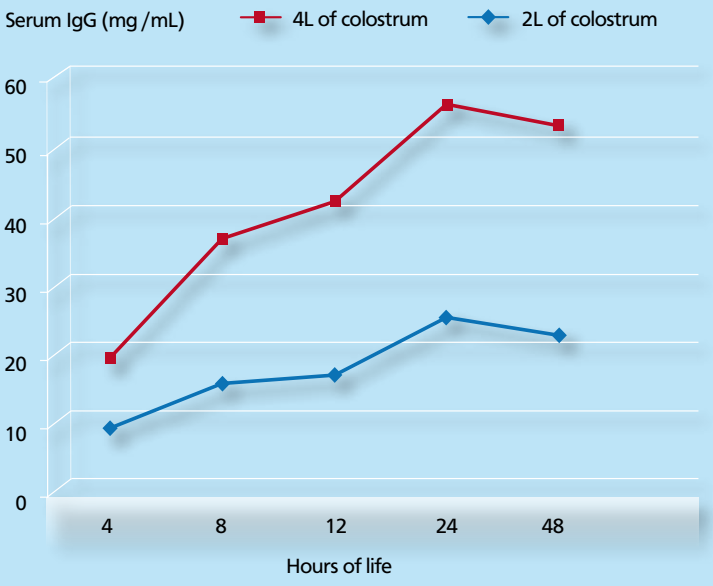
d.1.3. The calf

d.1.3.3. Volume of colostrum

- The best option: 4 L of colostrum at body temperature administered by a nipple bottle.

  Besser et al. [38] showed that only 36% of Holstein dams produced the necessary Ig level (>100 g Ig) in 1.89 L of colostrum.
  In contrast, 85% of them produced this level in 3.78 L

- If possible, continue with 2 L/12 h for at least 3 days as the colostral antibodies can neutralise the pathogens or toxins inside the intestines.



Serum IgG concentration in calves fed either 2 or 4 L of good-quality colostrum (60.1 mg of IgG/mL) at birth, and an additional 2 L at 12 hours of age [39]

The best option is to administer 4 L of good-quality colostrum (>60 mg Ig G7/ml) at birth.

d.1. Identifying good practices to ensure dam-to-calf immune transfer

d.1.3. The calf

d.1.3.4. Administration of colostrum

FRESH COLOSTRUM

Should be administered by the farmer:

- **TO ENSURE SUFFICIENT INTAKE OF COLOSTRAL ANTIBODIES:**
  Colostrum should be collected and administered with a nipple bottle. If the calf does not co-operate, a feeding tube can be used, though this will mean a delay of 2-3 hours for the colostrum to arrive in the abomasum, since the oesophageal groove will not work.

  When administration of colostrum by the dam or the farmer was evaluated [38] the percentage of calves in which passive immune transfer of Ig failed was
  - ✓ 61.4% of calves nursed by their dams
  - ✓ 19.3% of calves fed using a nipple bottle
  - ✓ 10.8% of calves fed using a tube

- **TO AVOID TRANSMISSION OF PATHOGENS:**
  Some pathogens can be transmited from dam to calf, so it is recommended not to leave them together for more than 1 hour after parturition.

To ensure colostrum intake, the farmer must collect colostrum and administer it to the calf with a nipple bottle

CAUTION...

Oral administration of any type of products before or with colostrum may disrupt the mechanism of immunoglobulin absorption

Administering colostrum using tube feeding is also a good option, but colostrum arrival to abomasum willl be delayed 2-3 hours.

d.2. Assessing the process

In order to monitor the farm situation register calf morbidity and mortality during rearing, as well as the causes of these problems and the treatments adopted.
This information will provide a picture of the farm situation, which can be compared to previously defined targets (see dairy rearing targets in the table).
In addition, we should periodically monitor passive immune transfer at the farm by sampling calf serum and colostrum quality, checking for immunoglobulins and contamination.
Periodic monitoring for environmental contamination is recommended.

Dairy calf health and performance targets [40,41]

Mortality (%)	0 - 24 hours	< 6
	1 - 60 days	< 3
	2 - 6 months	< 2
	6 - 24 months	< 1
Morbidity (%)	Diarrhoea 1 - 60 days	< 20
	Diarrhoea 2 - 6 months	< 5
	Diarrhoea 6-24 months	< 1

MORBIDITY AND MORTALITY DUE TO DIARRHOEA DURING REARING

EVALUATE WHETHER REAL FARM DATA ARE WITHIN THE TARGET

FAILURE OF DAM-TO-CALF PASSIVE TRANSFER OF Ig

EVALUATE CALF IMMUNOLOGICAL STATUS

EVALUATE COLOSTRUM QUALITY: Ig AND MICROBIOLOGY

EVALUATE ENVIRONMENTAL CONTAMINATION

EVALUATE BEDDING MATERIAL

d.2.1. Evaluating calf immunological status

Colostrum intake can be evaluated by quantifying immune transfer in blood:

Evaluation of immune transfer directly through IgG level and indirectly through STS

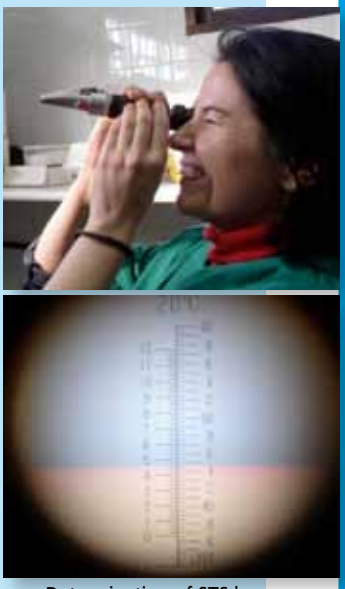
Parameter to evaluate immune transfer	Target value
IgG level	> 10.0 mg/mL
Serum total solids (STS)	> 5.5 g/dL

- **DIRECT DETERMINATION OF IgG LEVEL.**
  Plain blood samples from a minimum of 5 calves 2 to 5 days old should be sent to a laboratory within 24 hours of collection to measure IgG levels IgG should be higher than 10.0 mg/mL.

- **ESTIMATION OF SERUM IgG LEVEL.**
  Measurement of serum total solids (STS) by a hand-held refractometer in the serum of calves aged between 24 h and 7 days is simple, rapid and inexpensive and shows good correlation with serum IgG. However, results should be interpreted at the group level and not at the individual level.

  ✓ Good passive transfer has occurred if STS >5.5 g/dL [28]

  ✓ With good management, STS should reach values of 6-6.5 g/dL.



Determination of STS by refractometry

TIP... If we measure STS in the serum of 12 calves aged between 24 h and 7 days, 80% should have STS >5.5 g/dL when dam immune transfer is adequate. If more than 20% of calves are below the cutoff point → herd problem

TIP... Performing serum refractometry is the perfect time to make the farmer aware of the importance of good practices to prevent diarrhea!

CAUTION... Serum should be at 20 ºC for a more precise determination!

Example of STS sampling of colostrum intake and interpretation of results

Calves below 5.5 g/dl	Percentage (%)	Interpretation
1/12	8.3	No colostrum intake problem
2/12	16.7	Borderline
4/12	33.3	Failure of dam-to-calf passive transfer of Ig

d.2.2. Evaluating colostrum quality
d.2.2.1. Immunoglobulins

Colostrometer

There are large individual differences in colostrum Ig concentration. For example, in the Holstein breed, it varies between 9 and 186 g/L.

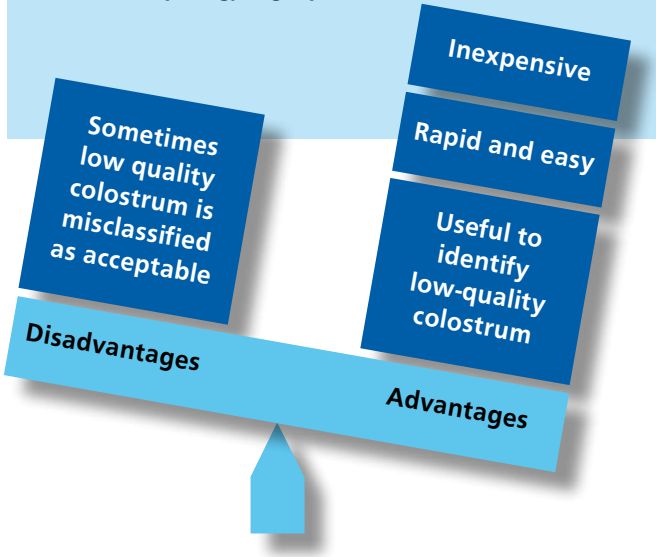
To evaluate colostrum quality:

- **MACROSCOPIC ASPECTS**, such as volume produced, intensity of colour or thickness; these aspects do not accurately indicate its quality
- **COLOSTROMETER**. Colostrum can be tested using a colostrometer, which evaluates the quality by estimating IgG concentration based on specific gravity.
- **Treshold value of specific gravity >1.050 (>50 g/L lg G)**

Colostrum appearance: the colostrum at the left looks yellower and thicker

TIP... Using the colostrometer is a perfect time to teach the farmer about the importance of farm management to prevent diarrhoea!

CAUTION... Colostrum should be at 20° C for a more precise determination!



Advantages and disadvantages of the colostrometer technique

Colostrometer measuring colostrum

d.2.2. Evaluating colostrum quality
d.2.2.2. Specific antibody levels

Antibody levels against rotavirus and coronavirus can be evaluated by neutralisation test or ELISA.
Suitable samples for test include colostrum or milk.

Expected titers for antibody levels in colostrum following vaccination

Days post calving	Avarage titer	Lower limit acceptable
0 - 1	10240	5120
1 - 2	2560	1280
2 - 3	1280	640
3 - 4	640	320
4 - 5	320	160

Calves fed adequate levels of colostrum at the correct time would be expected to have IgG values in the range of 10-25 mg/ml

d.2.2. Evaluating colostrum quality
d.2.2.3. Microbiology

- **High bacterial contamination not only interferes with Ig absorption, but also has a direct pathogenic effect.**
- **Colostrum contamination can be assessed by two parameters:**

Parameters to evaluate colostrum contamination

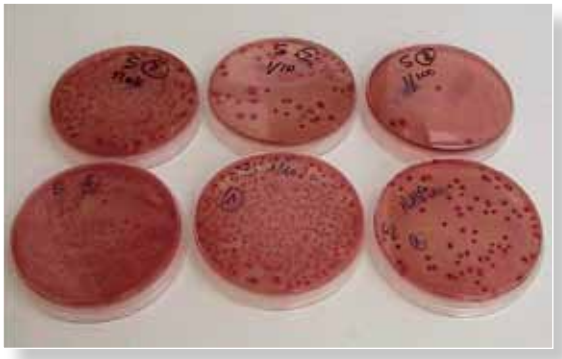
Parameter	Target value
Total bacteria count	<100,000 CFU/mL
Total coliform count	<10,000 CFU/mL

- **This is specially important if the colostrum is going to be frozen or refrigerated.**

NEVER...

Store colostrum at room temperature to avoid bacterial multiplication

Colostrum storage at room temperature will increase bacteria count



Bacteria count

d.2.3. Evaluating environmental contamination

To evaluate contamination of the environment, we can submit samples of bedding material for bacteriological counts and *Salmonella spp.* culture.

HOW TO TAKE THOSE SAMPLES?

Samples should be taken with gloved hands from the perimeter of the pen in each of the four quadrants and from the centre of the pen, specifically avoiding the sampling of faecal material.

WHICH SAMPLES CAN WE TAKE?

Take samples of different housing facilities such as the maternity pen, communal hutch, individual pens, etc.

Although standards for the level of bacterial contamination in calf bedding have not yet been established, farm results can be evaluated against targets based on the level of risk cited for environmental mastitis [42].

Parameters to evaluate bedding contamination

Sample source	Total coliform count	Total bacteria count	*Salmonella spp.*
Goal for a clean pen	<1,000 CFU/mL	<5,000 CFU/mL	Neg
Goal for an occupied pen	<500,000 CFU/mL	<2,000,000 CFU/mL	Neg

E. Prevention and treatment

e.1. Biosecurity

e.1.1. General aspects

1) LIMIT DAM-TO-CALF TRANSMISSION

In many cases, calves become infected by their dams or other cows by direct contact or indirectly as a result of environmental contamination

Immunosuppression in periparturient cows causes shedding of large numbers of pathogens in faeces or respiratory secretions

MATERNITY AREA

- *Calving area should be clean and disinfected*
- *Avoid multiple-cow pens*
- *Separate the calf from the dam as soon as possible (<1 hour)*

COLOSTRUM AND MILK

When infectious diseases transmitted through colostrum (such as Johne's disease) are present in the herd, pasteurisation of colostrum and use of milk replacers may be a solution

2) LIMIT CALF-TO-CALF TRANSMISSION

By direct contact when separation is insufficient, or by indirect contact through contaminated shared material

- *Housing of calves in individual calf pens Pens cleaned, and disinfected between successive calves*
- *Cleaning and disinfection of feeding and other equipment on a daily basis*
- *Caregivers:*
  - *Handle calves from youngest to oldest*
  - *Wear gloves to assist sick calves (change after each calf)*
  - *Clean and disinfect clothes and boots after handling sick calves*

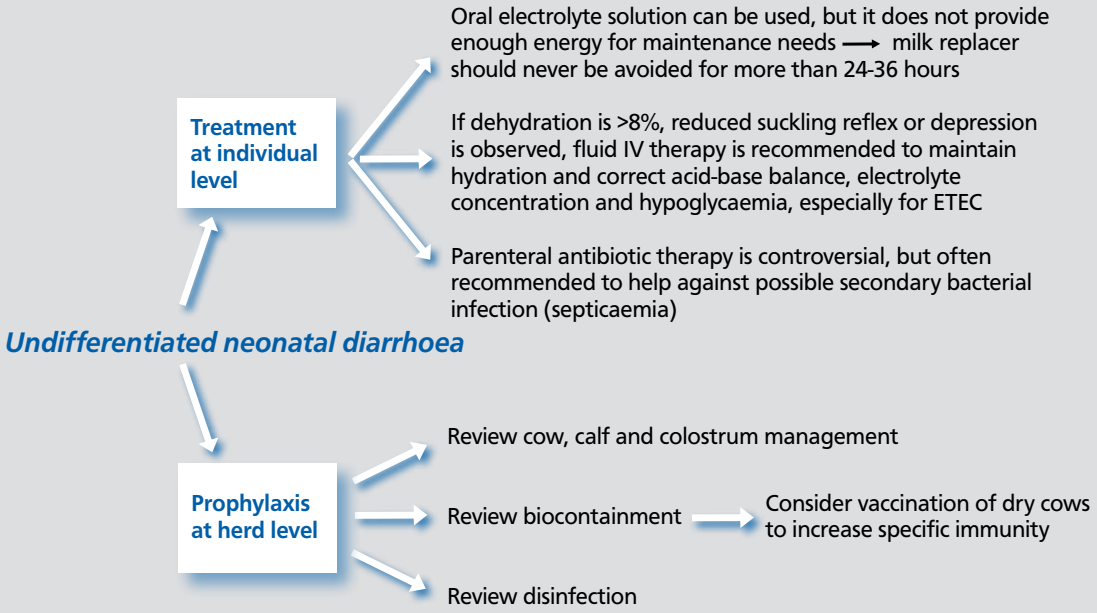
3) KEEP RECORDS OF HEALTH DATA

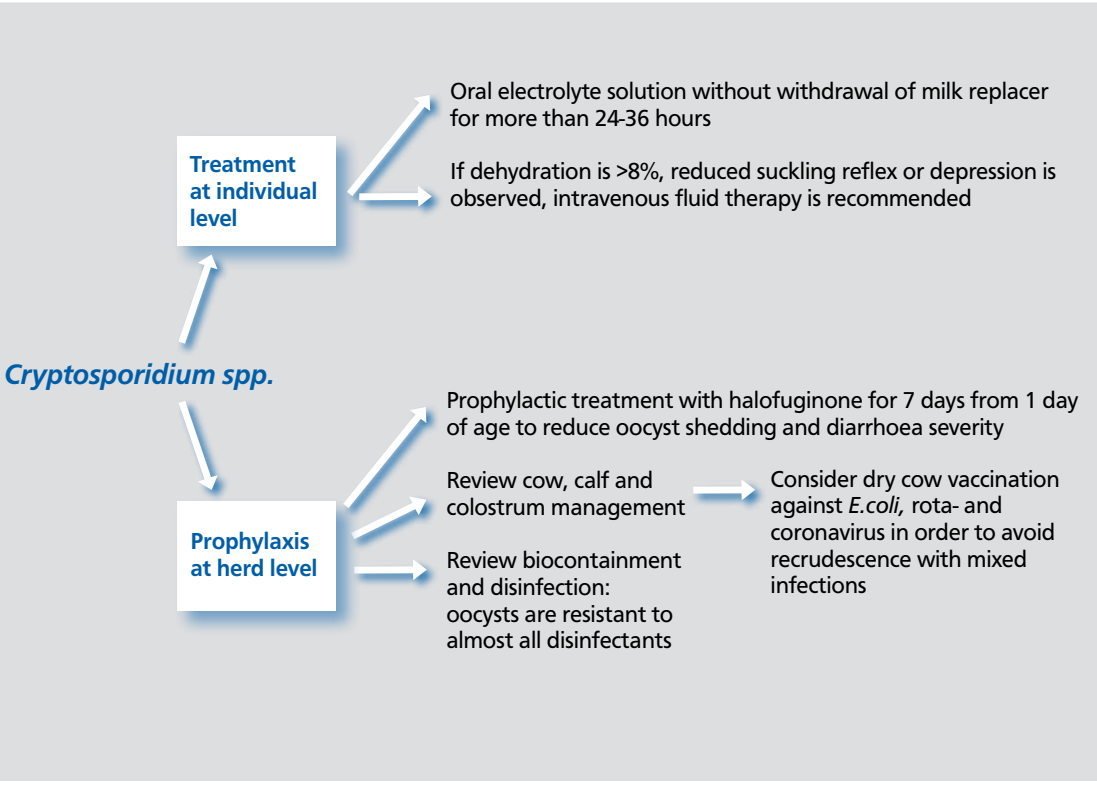
Regular data monitoring allows prompt recognition of problems

Review herd status other diseases (e.g. Bovine Viral Diarrhoea)

e.1. Biosecurity

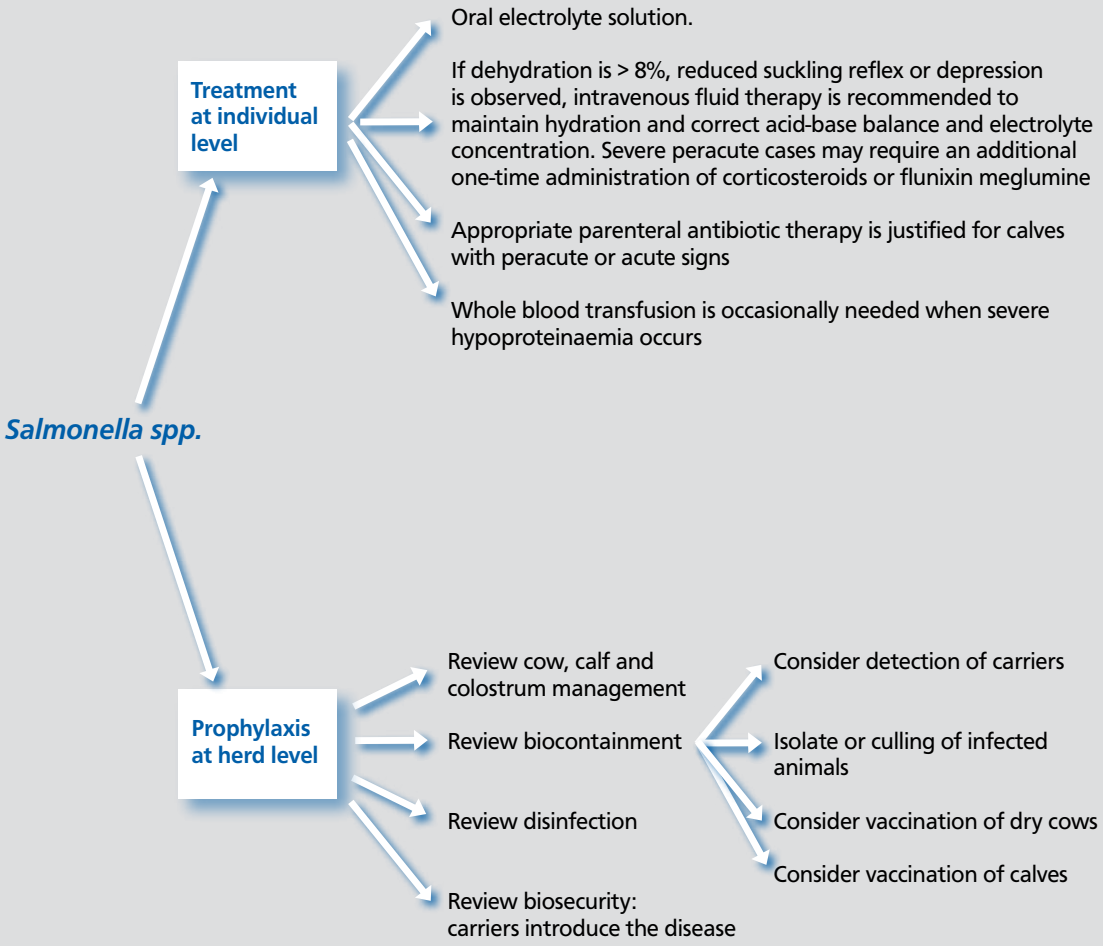
e.1.2. Pathogen by pathogen





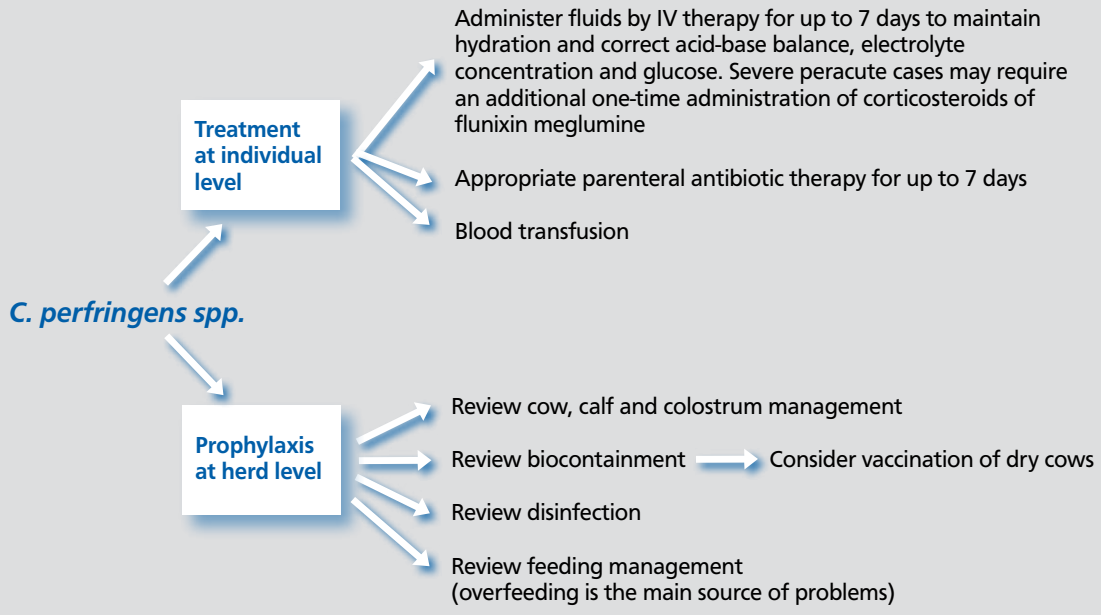
e.1. Biosecurity

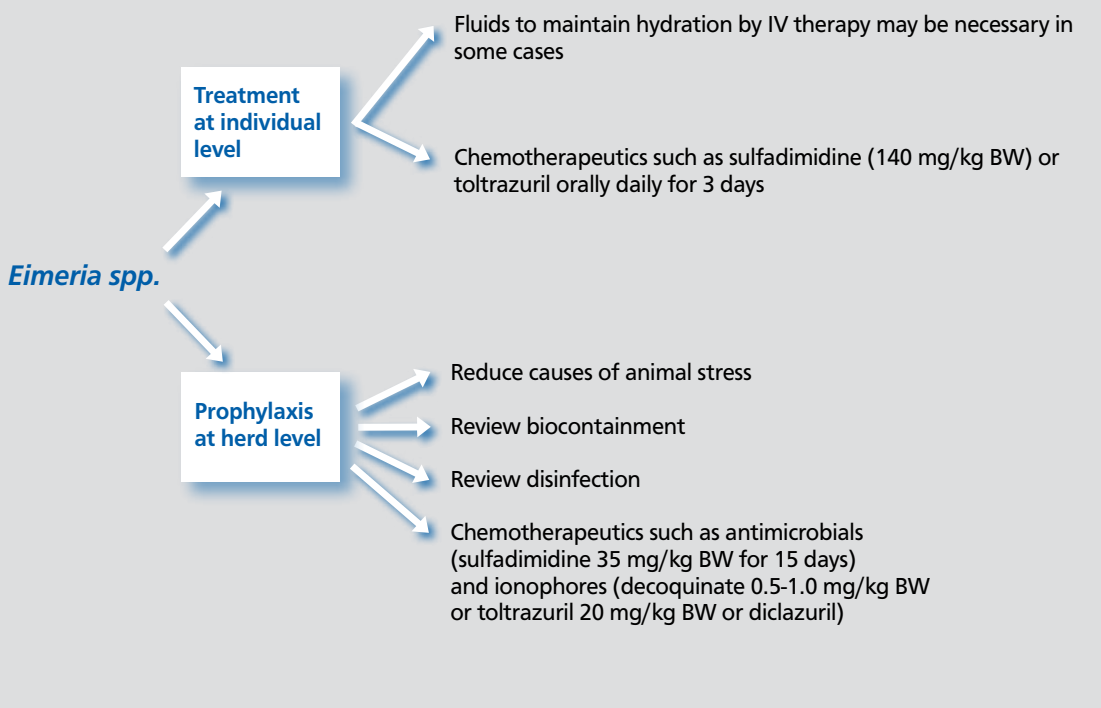
e.1.2. Pathogen by pathogen



e.1. Biosecurity

e.1.2. Pathogen by pathogen





e.2.1. Enhancing colostrum quality

DAM VACCINATION

It has been shown that vaccines administered to a cow before calving increase colostral antibodies specifically against those antigens. This has been best demonstrated in the case of vaccines against neonatal diarrhoea pathogens.
Vaccination should be done 3-12 weeks before the expected calving date.

Implementation of vaccination protocols of dry cows:

- Rotavirus – coronavirus – *E. coli*
- *Salmonella spp.*
- *C. perfringens*

→ Reduces pathogen excretion by the dam at parturition

→ Increases specific antibodies against those pathogens we vaccinate for

Vaccinating a dry cow

TIP...

Be aware of the need for colostrum management on the farm!

Although dam vaccination enhances colostrum quality, if it is not correctly administrated to the calves, diarrhoea problems will continue in the herd!

ADMINISTERING VITAMIN E AND SELENIUM TO THE DAM

SELENIUM and VITAMIN E SUPPLEMENTATION by injection or by supplementing the dry cow ration may be worthwhile, since it increases colostrum volume above that of dams deficient in these additives [44].

Keeping herd performance on track means preventing and controlling neonatal diarrhea.

Evaluating herd and farm management practices, properly diagnosing pathogens and analyzing colostrum quality and intake are essential steps in resolving this costly problem. Selecting the right prevention / treatment protocol is also critical to success.

The five step program, that is summarized on the following pages can be a useful tool to improve neonatal health on problem farms.

A possible protocol that can be used in practice includes the following steps:

Step 1- Anamnesis

In the process of investigating a case of neonatal diarrhoea on a farm, a thorough discussion with the cattle producer about calf management and type of animals affected can already identify a list of possible causes of the diarrhoea problem.

Key data: age of the animals affected, colostrum management, calf feeding protocol, housing conditions, previous farm disease history and veterinary Health Plan

Step 2- Faecal sampling

Enteric conditions caused by infectious microorganisms can be diagnosed from fresh faecal samples. When sampling a herd the following should be considered:

- Sample a group of at least five affected animals
- Collect faecal samples from the animal and not from the floor

On-site diagnosis is an easy and quick option for Rotavirus, Coronavirus, *E. coli* and *C. parvum.*

Step 3- Evaluating the colostrum intake

Colostral antibodies provide local protection in the gut of the calf but a portion is also absorbed into the blood stream. The capacity for absorption of antibodies is high during the first hours after birth and disappears once the calf is 24 hours old.
The quality of the colostrum feeding regime can be evaluated by measuring the level of IgG in blood. Values of less than 10 g/l are indicative of inadequate colostrum intake.

Step 4- Measuring the quality of the colostrum

A low level of calf serum Ig can be the result of receiving poor-quality colostrum. Colostrum quality can be measured using a colostrometer. If colostrum does not contain sufficient antibody levels, action can be taken. Specific colostral antibodies can be boosted by vaccination.

Step 5- Define and implement a prevention/treatment protocol

Actions following the diagnosis of neonatal diarrhoea on a farm are at three levels.

- Treatment of affected animals: Rehydration (IV/oral), antibiotic and/or *C. parvum* treatment, NSAIDs
- Colostrum management: Corrective measures must be taken if problems are identified in the administration of colostrum to neonatal calves.
- Prevention: Implementation of vaccination protocols with Rotavec® Corona and preventive use of Halocur® for the control of *C. parvum*.

References

[1] USDA (2007) Dairy, Part I: Reference of Dairy Cattle Health and Management Practices in the United States, USDA-APHISVS,CEAH, #N480.1007. National Animal Health Monitoring System, Fort Collins, CO.

[2] USDA (1997) Beef, Part II: Reference of Beef Cow-Calf Health & Health Management Practices. USDA-APHISVS,CEAH. National Animal Health Monitoring System, Fort Collins, CO.

[3] Couture Y, Major RR (1989) Resultats sur la mortalite des veaux de type boucherie de la region Abitibi-Temiscamingue. Les principaux problemes de sante chez le veau. Quebec: Ministere de l'Agriculture, des Pecheries et de l'Alimentation du Quebec.

[[4] Heinrichs AJ, Radostits OM (2001) Health and production management of dairy calves and replacement heifers. In: Radostits OM, editor: Herd Health Food Animal Production Medicine 3rd ed. Philadelphia: WB Saunders. pp. 333-473.

[5] Virtala AMK, Mechor GD, Gröhn YT, Hollins NE (1996) Morbidity from non respiratory diseases and mortality in dairy heifers during the first three months of life. J Am Vet Med Assoc; 208 (12): 2043-6.

[6] Wells SJ, Garber LP, Hill GW (1996) Health status of preweaned dairy heifers in the United States Prev Vet Med; 29: 185- 99.

[7] Bendali F (2009) Risk factors for neonatal diarrhea in beef calves. In: Procc. Neonatal health in calves. Comprehensive solutions for complex enteric disorders. Barcelona (Spain), 8-9 June 2009.

[8] Schmoldt P, Bilnger U, Pongk J, Kleiner W, Brade W, Motsch T,Kaphengst P, Rotermund H (1979) Einfluss van Erkrankungen auf die Zuwachsleistung sowie die Nahrstoffaufnahme und-verwertung von Trankkalbern. [Effects of diseases on growth, nutrient intake, and nutrient conversion comparison in drinking calf.] Monatsh Veterinaemed; 34: 95.

[9] Waltner-Toews D, Martin SW, Meek AH (1986) The effect of early calfhood health status on survivorship and age at first calving. Can J Vet Res; 50(3): 314-7.

[10] Correa MT, Curtis CR, Erb HN, White ME. (1988) Effect of calfhood morbidity on age at first calving in New York Holstein herds. Prev Vet Med; 6: 253-62.

[11] Hoffman PC, Funk DA (1992) Applied Dynamics of Dairy Replacement Growth and Management. J Dairy Sci; 75(9): 2504-16.

[12] Wittum TE, Salman MD, Odde KG, Mortimer RG, King ME (1993) Causes and costs of calf mortality in Colorado beef herds participating in the National Animal Health Monitoring System. J Am Vet Med Assoc; 203: 232–6.

[13] Ganaba RC, Bigras-Poulin M, Bdlanger D, Couture Y (1995) Description of cow-calf productivity in Northwestern Quebec and path models for calf mortality and growth Prev Vet Med; 24 (3): l-42.

[14] Virtala AMK, Mechor GD, Gröhn YT (1996) The effect of calfhood diseases on growth of female dairy calves during the first 3 months of life in New York State. J Dairy Sci; 79: 1040-9.

[15] Donovan GA, Dohoo IR, Montgomery DM, Bennett FL (1998) Calf and disease factors affecting growth in female Holstein calves in Florida, USA. Prev Vet Med; 33: 1 - 10.

[16] Gunn GJ and Stott AW (1998) A comparison of economic losses due to calf enteritis and calf pneumonia in Scottish herds. In: Caple IW, editor. Procc XX World Buiatrics Congress. jul 6-10; Sidney, Australia: pp. 357-60.

[17] Svensson C, Lundborg K, Emanuelson U, Olsson SO (2003) Morbidity in Swedish dairy calves from birth to 90 days of age and individual calf-level risk factors for infectious diseases. Prev Vet Med; 58: 179-97.

[18] Svensson C, Hultgren J (2008) Associations Between Housing, Management, and Morbidity During Rearing and Subsequent First-Lactation Milk Production of Dairy Cows in Southwest Sweden. J Dairy Sci; 91: 1510–18.

[19] Gulliksen SM, Lie KI, Løken T, Osterås O (2009) Calf mortality in Norwegian dairy herds. J Dairy Sci; 92(6): 2782-95.

[20] Soberon F, Raffrenato E, Everett RW, van Amburg ME (2009) Early life management and long term productivity of dairy calves. Procc Joint Annual Meeting ADSA-CSAS-ASAS p. 130.

[21] Boileau MJ, Kapil S (2010) Bovine coronavirus associated syndromes Veterinary Clinics of North America: Food Animal Practice Emerging, Reemerging, and Persistent Infectious Diseases of Cattle; 26(1): 123-46.

[22] Olson, M.E., O'Handley, R.M., Ralston, B.J., McAllister, T.A. and Thompson, R.C.A. (2004) Update on Cryptosporidium and Giardia infections in cattle. Trends in Parasitology; 20(4): 185-91.

[23] Bartels CJM, Holzhauer M, Jorritsma R, Swart WAJM, Lam TJGM (2010) Prevalence, prediction and risk factors of enteropathogens in normal and non-normal faeces of young Dutch dairy calves. Prev Vet Med; 93(2-3): 162-9.
[24] Radostits OM, Gay CC, Hinchcliff KW, Constable PD (2007) Acute undifferentiated diarrhea of newborn farm animals. In: Veterinary medicine. A text book of the diseases of cattle, sheep, pigs, goats and horses. W.B. Saunders Co. Philadelphia, USA. ISBN: 978-0-7020-2777-2. Pp. 837-51.
[25] Bouda J, Medina CM, Núñez OL, Candanosa AE, Paasch ML, Lemus V (1009) Microbiology and clinicopathological findings in calves with acute diarrhea and suggested intravenous fluid therapy. Proceedings of XX World Buiatrics Congress 1998, Jul 6-10; Sydney, Australia; 1: 375-9.
[26] García A, Ruiz-Santa-Quiteria JA, Orden JA, Cid D, Sanz R, Gómez-Bautista M, de la Fuente R (2000) Rotavirus and concurrent infections with other enteropathogens in neonatal diarrheic dairy calves in Spain. Comp Immunol MicrobiolInfect Dis; 23: 175-83.
[27] De la Fuente R, Luzón M, Ruiz-Santa-Quiteria JA, García A, Cid D, Orden JA, García S, Sanz R, Gómez-Bautista M (1999) Cryptosporidium and concurrent infections with other major enterophatogens in 1 to 30-day-old diarrheic dairy calves in central Spain. Vet Parasitol; 80: 179-85.
[28] McGuirk S (2008) Disease management of dairy calves and heifers. Vet Clin North Am Food Anim Pract; 24(1): 139-53.
[29] Lorenz I, Vogt S. (2006) Investigations on the association of D-lactate blood concentrations with the outcome of therapy of acidosis, and with posture and demeanor in young calves with diarrhoea. J Vet Med A; 53: 490-4.
[30] Schelcher F, Macillaud S, Braun JP, Contrepois M, Valarcher JF, Navetat H (1998) Metabolic acidosis without dehydration and no minimal diarrhoea in suckler calves is caused by hyper D-lactatemia. Proceedings of XX World Buiatrics Congress 1998, jul 6-10; Sydney, Australia, 1: 371-4.
[31] Geishauer T, Thünker B (1997) Metabolische Azidose bei neugeborenen Kälbern mit Durchfall: Abschätzung an Saugreflex oder Stehvermögen. Prakt Tierarzt; 78: 600-5.
[32] Philippeau C, Goncalves S, Julliand V. (2004) Enterotoxaemia in Charolais cattle in Burgundy: new decision support tools to improve bacterial diagnosis and hypotheses of risk factors, Renc Rech Ruminants. 11: 321-4.
[33] Grusenmeyer DJ, Ryan CM, Galton DM, Overton TR (2006) Shortening the dry period from 60 to 40 days does not affect colostrum quality but decreases colostrum yield by Holstein cows J Anim Sci; 84 (Suppl, 1): 336.
[34] Grummer RR, Rastani RR (2004) Why Reevaluate Dry Period Length? J. Dairy Sci; 87: 77-85.
[35] Moore M, Tyler JW, Chigerwe M, Dawes ME, Middleton JR (2005) Effect of delayed colostrum collection on colostral IgG concentration in dairy cows. J Am Vet Med Assoc; 226(8): 1375-7.
[36] Cortese VS (2009) Neonatal immunology. Vet Clin North Am Food Anim Pract; 25(1): 221-7.
[37] Gooden S (2008) Colostrum Management for Dairy Calves; Vet Clin North Am Food Anim Pract; 24(1): 19-39.
[38] Besser TE, Gay CC Pritchett L (1991) Comparison of three methods of feeding colostrum to dairy calves. J Am Vet Med Assoc; 198(3): 419-22.
[39] Morin DE, McCoy GC, Hurley WL (1997) Effects of quality, quantity, and timing of colostrum feeding and addition of a dried colostrum supplement on immunoglobulin G1 bsorption in Holstein bull calves. J Dairy Sci; 80: 747–53.
[40] Quigley JD, Nyabadza CST, Benedictus G, Brand A (1996) Monitoring replacement rearing: objectives and materials and methods. In: Herd health and production management in dairy practice: Eds. A. Brand, J.P.T.M. Noordhuizen and Y. Schukken, Wageningen Press, PO Box 42, 6700 AA Wageningen, The Netherlands; pp: 75-170.
[41] Bailey TL, Murphy JM (1999) Dairy heifer development and monitoring. In: Howard and Smith, Current Veterinary Therapy 4, Food Animal Practice, WB Saunders Co., Philadelphia, Pen. fourth ed; pp. 86-93.
[42] McGuirk S. (2003) Solving Calf Morbidity and Mortality Problems. American Association of Bovine Practitioners 36th Annual Conference, September 15-17, - Columbus, OH.
[43] González JV and Astiz S (2005) Actuación Veterinaria. In: Diarreas en el ternero neonato. Ed Schering Plough AH, Zaragoza ISBN: 84-689-2874-7; pp. 145-60.
[44] Lacetera N, Bernabucci U, Ronchi B, Nardone A (1996) Effects of selenium and vitamin E administration during a late stage of pregnancy on colostrum and milk production in dairy cows, and on passive immunity and growth of their offspring. Am J Vet Res; 57(12):1776-80.

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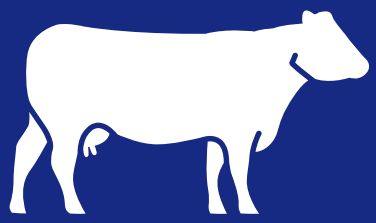
EARLY CARE, LIFE LONG GAINS



Intervet International bv
PO Box 31, 5830 AA Boxmeer
The Netherlands
Phone: +31 (0)485 587600
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